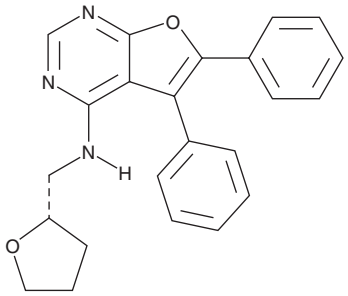
PRODUCT INFORMATION

AIM 100

Item No. 29487

CAS Registry No.:	873305-35-2
Formal Name:	5,6-diphenyl-N-[[(2S)-tetrahydro-2-furanyl]methyl]-furo[2,3-d]pyrimidin-4-amine
MF:	$C_{23}H_{21}N_3O_2$
FW:	371.4
Purity:	≥98%
UV/Vis.:	λ_{max}: 320 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

AIM 100 is supplied as a crystalline solid. A stock solution may be made by dissolving the AIM 100 in the solvent of choice, which should be purged with an inert gas. AIM 100 is soluble in organic solvents such as ethanol, DMSO, and dimethyl formamide (DMF). The solubility of AIM 100 in ethanol and DMSO is approximately 10 mg/ml and approximately 16 mg/ml in DMF.

AIM 100 is sparingly soluble in aqueous buffers. For maximum solubility in aqueous buffers, AIM 100 should first be dissolved in DMF and then diluted with the aqueous buffer of choice. AIM 100 has a solubility of approximately 0.2 mg/ml in a 1:4 solution of DMF:PBS (pH 7.2) using this method. We do not recommend storing the aqueous solution for more than one day.

Description

AIM 100 is an inhibitor of activated Cdc42 kinase 1/tyrosine non-receptor kinase 2 (ACK1/TNK2; IC_{50} = 21.58 nM).[1] It is selective for ACK1/TNK2 over Abl1, BTK, LCK, and LYN (IC_{50}s = 705.9, 871.7, 432.3, and 346.7 nM, respectively), as well as a panel of 25 additional kinases. AIM 100 (1 µM) inhibits EGF-induced increases in ataxia-telangiectasia mutated kinase (ATM) protein levels, ACK1/TNK2 and androgen receptor phosphorylation, and $pTyr^{267}$-androgen receptor binding to the ATM enhancer in LAPC4 cells. It inhibits the growth of LNCaP and LAPC4 cells in a concentration-dependent manner and induces cell cycle arrest at the G_0/G_1 phase when used at a concentration of 3 µM.[2] AIM 100 (4 mg/kg) enhances radiation-induced tumor growth reduction in an LNCaP-caAck castration-resistant prostate cancer (CRPC) mouse xenograft model.[1]

References

1. Mahajan, K., Coppola, D., Rawal, B., *et al.* Ack1-mediated androgen receptor phosphorylation modulates radiation resistance in castration-resistant prostate cancer. *J. Biol. Chem.* **287(26)**, 22112-22122 (2012).
2. Mahajan, K., Challa, S., Coppola, D., *et al.* Effect of Ack1 tyrosine kinase inhibitor on ligand-independent androgen receptor activity. *Prostate* **70(12)**, 1274-1285 (2010).

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