产品名称：**CYC116**
产品别名：**CYC-116**

生物活性：		
Description	CYC-116 is a potent aurora A and aurora B inhibitor with K_is of 8 and 9 nM, respectively.	
IC_{50} & Target [1]	Aurora A	Aurora B
	8 nM (Ki)	9.2 nM (Ki)
In Vitro	CYC-116 also inhibits VEGFR2, Src, Lck AND FLT3 with with Kis of 44, 82, 280, 44 nM, respectively. CYC-116 may have broad-spectrum antitumor activity. CYC-116 shows potent antiproliferative activity against cancer cell lines with with IC50s of 0.599, 0.59, 0.241, 0.34, 0.725, 1.375, 0.471, 0.034, 0.372, 0.681, 0.151, 1.626, 0.775, 0.308, 0.110, 0.09 for MCF7, HeLa, Colo205, HCT-116, HT29, K562, CCRF-CEM, MV4-11, HL60, NCI-H460, A2780, BxPC3, HuPT4, Mia-Paca-2, Saos-2, Messa cells. Treatment with 1.25 μM CYC-116 for 7 h results in complete inhibition of histone H3 phosphorylation in HeLa cell lysates[1].	
In Vivo	Oral administration of CYC-116 at dose levels of 75 and 100 mg/kg q.d. causes tumor growth delays of 2.3 and 5.8 days, which translates into specific growth delays of 0.32 and 0.81, respectively. The mean relative tumor volumes of mice receiving CYC-116 at both dose levels are less than those of vehicle-treated mice for the duration of the study period. At 100 mg/kg po q.d., the reduction in growth is statistically significant on days 6 and 9[1].	
Solvent&Solubility	***In Vitro:*** **DMSO : < 1 mg/mL** (insoluble or slightly soluble)	
References	[1]. Wang S, et al. Discovery of N-phenyl-4-(thiazol-5-yl)pyrimidin-2-amine aurora kinase inhibitors. J Med Chem. 2010 Jun 10;53(11):4367-78.	

实验参考：	
Cell Assay	CYC-116 is prepared in DMSO and diluted in cell medium[1].
Animal Administration	Mice: Mice implanted intraperitoneally with P388/0 cells are treated with CYC-116, and the antitumor activity is measured as an increase in lifespan of the treated animals versus the vehicle control group[1].
Kinase Assay	Aurora A kinase assays are performed using a 25 μL reaction volume (25 mM β-glycerophosphate, 20 mM Tris/HCl, pH 7.5, 5 mM EGTA, 1 mM DTT, 1 mM Na_3VO_4, 10 μg of kemptide (peptide substrate)), and recombinant aurora A kinase is diluted in 20 mM Tris/HCl, pH 8, containing 0.5 mg/mL BSA, 2.5% glycerol, and 0.006% Brij-35. Reactions are started by the addition of 5 μL Mg/ATP mix (15 mM $MgCl_4$, 100 μM ATP, with 18.5 kBq γ-^{32}P-ATP per well) and incubated at 30°C for 30 min before terminating by the addition of 25 μL of 75 mM H_3PO_4. Aurora B kinase assays are performed as for aurora A except that prior to use, aurora B is activated in a separate reaction at 30°C for 60 min with inner centromeres protein [1].
References	[1]. Wang S, et al. Discovery of N-phenyl-4-(thiazol-5-yl)pyrimidin-2-amine aurora kinase inhibitors. J Med Chem. 2010 Jun 10;53(11):4367-78.