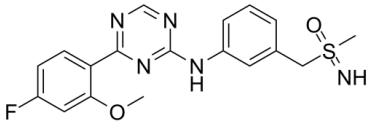
Data Sheet

Product Name:	Atuveciclib Racemate
Cat. No.:	CS-6307
CAS No.:	1414943-88-6
Molecular Formula:	C18H18FN5O2S
Molecular Weight:	387.43
Target:	CDK
Pathway:	Cell Cycle/DNA Damage
Solubility:	10 mM in DMSO



BIOLOGICAL ACTIVITY:

Atuveciclib Racemate (BAY-1143572 Racemate) is the racemate mixture of Atuveciclib. Atuveciclib is a potent and highly selective, oral **P-TEFb/CDK9** inhibitor which supresses **CDK9/CycT1** with an **IC_{50}** of 13 nM. IC50 & Target: IC50: 385 nM (AML cell lines)[1] **In Vitro:** Atuveciclib (BAY-1143572) inhibits the proliferation of 7 MLL-rearrangements positive and negative AML cell lines with a median IC_{50} of 385 nM (range 230-1100 nM) and induces apoptosis[1]. Atuveciclib (BAY-1143572) has potent and highly selective PTEFb-kinase inhibitory activity in the low nanomolar range against PTEFb/CDK9 and an at least 50-fold selectivity against other CDKs. Atuveciclib (BAY-1143572) shows a favorable selectivity against a panel of non-CDK kinases. It shows broad antiproliferative activity against a panel of tumor cell lines with sub-micromolar IC_{50} values. The concentration-dependent inhibition of the phosphorylation of the RNA polymerase II and downstream reduction of MYC mRNA and protein levels is observed[2]. **In Vivo:** Atuveciclib (BAY-1143572) exhibits single agent efficacy at tolerated doses in 4 out of 5 AML xenograft tumor models in mice and in 2 out of 2 AML xenograft tumor models in rats upon once daily oral administration. Partial or even complete remissions could be achieved in several models[1].The inhibition of MYC mRNA is also observed in blood cells of Atuveciclib (BAY-1143572)-treated rats indicating the potential clinical utility of MYC in blood cells as a pharmacodynamic marker in clinical development. The in vivo efficacy of Atuveciclib (BAY-1143572) is significantly enhanced in combination with several chemotherapeutics in different solid tumor models[2].

References:

[1]. Scholz A, et al. BAY 1143572, a first-in-class, highly selective, potent and orally available inhibitor of PTEFb/CDK9 currently in Phase I, shows convincing anti-tumor activity in preclinical models of acute myeloid leukemia (AML). [abstract]. In: Proceedings of the 107th Annual Meeting of the American Association for Cancer Research; 2016 Apr 16-20; New Orleans, LA. Philadelphia (PA): AACR; Cancer Res 2016;76(14 Suppl):Abstract nr 3022.

[2]. Scholz A, et al. BAY 1143572: A first-in-class, highly selective, potent and orally available inhibitor of PTEFb/CDK9 currently in Phase I, inhibits MYC and shows convincing anti-tumor activity in multiple xenograft models by the induction of apoptosis. [abstract]. In: Proceedings of the 106th Annual Meeting of the American Association for Cancer Research; 2015 Apr 18-22; Philadelphia, PA. Philadelphia (PA): AACR; Cancer Res 2015;75(15 Suppl):Abstract nr DDT02-02. doi:10.1158/1538-7445.AM2015-DDT02-02

CAIndexNames:

1,3,5-Triazin-2-amine, 4-(4-fluoro-2-methoxyphenyl)-N-[3-[(S-methylsulfonimidoyl)methyl]phenyl]-

SMILES:

N=S(CC1=CC(NC2=NC(C3=CC=C(F)C=C3OC)=NC=N2)=CC=C1)(C)=O

Caution: Product has not been fully validated for medical applications. For research use only.

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