Product Data Sheet

Product Name: 360A
Cat. No.: GC15389

Chemical Properties

Cas. No.	794458-56-3		
Chemical Name	3,3'-((pyridine-2,6-dicarbonyl)bis(azanediyl))bis(1-methylquinolin-1-ium)		
SMILES	CN1=C2C=CC=CC2=C/C(C=1)=[NH+]/C(C3=NC(C(/[NH+]=C4C=C5C=CC=CC5=N(C)=C/4)=O)=CC=C3)=O		
Formula	$C_{27}H_{23}N_5O_2$ 2+	M.Wt	449.5
Solubility	Soluble in DMSO	Storage	Store at -20°C
General tips	For obtaining a higher solubility , please warm the tube at 37 ℃ and shake it in the ultrasonic bath for a while.Stock solution can be stored below -20℃ for several months.		
Shipping Condition	Evaluation sample solution : ship with blue ice All other available size: ship with RT , or blue ice upon request.		
Structure	360A		

Protocol

Cell experiment:

The cell proliferation reagent WST-1 assay is performed. In brief, cells are seeded at various densities, depending on cell type (0.25-4 × 103 cells/well in 100 μL complete medium), in 96-well culture plates and treated with various concentrations (0.1-20 μM) of 360A or the corresponding concentrations of DMSO (control wells) for 3 or 7 days at 37°C in an atmosphere containing 5% CO2. For 7-day assays, the medium is changed on day 3. Experiments are performed in triplicate[1].

Tel: (909) 407-4943 Fax: (626) 353-8530 E-mail: tech@glpbio.com
Address: 10292 Central Ave. #205, Montclair, CA, USA

References:

[1]. Pennarun G, et al. Apoptosis related to telomere instability and cell cycle alterations in human glioma cells treated by new highly selective G-quadruplex ligands. Oncogene. 2005 Apr 21;24(18):2917-28.
[2]. Gauthier LR, et al. Rad51 and DNA-PKcs are involved in the generation of specific telomere aberrations induced by the quadruplex ligand 360A that impair mitotic cell progression and lead to cell death. Cell Mol Life Sci. 2012 Feb;69(4):629-40.

Background

Description: IC50 Value: N/A 360A is a 2,6-pyridine-dicarboxamide derivative displaying strong affinity and selectivity for G-quadruplex structures and selective telomerase inhibition in vitro assays. 360A is a G-quadruplex ligand, which can influence the consequence of G-quadruplex formation and/or stabilization. in vitro: We found a S-phase accumulation in ATM-proficient, but not in ATM-deficient EBV-lymphocytes treated with 360A before induction of cell death. However, ATM status did not modify cell cycle distribution in 360A-treated SV40-fibroblasts and HeLa cells compared to DMSO treated controls [1]. DNA-PKcs-dependent NHEJ was responsible for sister telomere fusions as a direct consequence of G-quadruplex formation and/or stabilization induced by 360A on parental telomere G strands. NHEJ and HR activation at telomeres altered mitotic progression in treated cells [2]. This compound was shown to display a potent affinity and selectivity for telomeric G-quadruplex DNA over duplex DNA and to induce delayed

growth inhibition in HT1080 tumor cell line [3]. in vivo: N/A Clinical trial: N/A

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