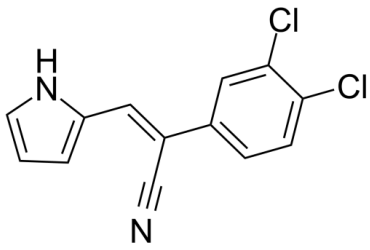
Data Sheet

Product Name:	ANI-7
Cat. No.:	CS-0063722
CAS No.:	931417-26-4
Molecular Formula:	$C_{13}H_8Cl_2N_2$
Molecular Weight:	263.12
Target:	Aryl Hydrocarbon Receptor; Checkpoint Kinase (Chk)
Pathway:	Cell Cycle/DNA Damage; Immunology/Inflammation
Solubility:	DMSO : 20.83 mg/mL (79.17 mM; Need ultrasonic)



BIOLOGICAL ACTIVITY:

ANI-7 is an activator of **aryl hydrocarbon receptor (AhR)** pathway. ANI-7 inhibits the growth of multiple cancer cells, and potently and selectively inhibits the growth of MCF-7 breast cancer cells with a GI_{50} of 0.56 μM. ANI-7 induces CYP1-metabolizing mono-oxygenases by activating **AhR** pathway, and also induces DNA damage, **checkpoint Kinase 2 (Chk2)** activation, S-phase cell cycle arrest, and cell death in sensitive breast cancer cell lines[1][2][3]. IC50 & Target: Aryl hydrocarbon receptor (AhR) pathway[1] **In Vitro:** ANI-7 (2.5 μM; 24 hours; MCF10A and MDA-MB-468 cells) treatment induces significant S-phase and G2 + M-phase cell cycle arrest within 24 hours of treatment in MDA-MB-468 cells, and negligible effect in normal breast MCF10A cells[1].

ANI-7 (2 μM; 12-24 hours; MDA-MB-468 cells) treatment results in a significant increase in the content and phosphorylation of CHK2, and induces a significant increase in H2AXɣ in MDA-MB-468 cells, indicative of DNA double-strand damage[1].

Inhibition of the AhR pathway ameliorates the effects of ANI-7. ANI-7 activates XRE activity and expression of the AhR and CYP1 members[1].

Comparisons of the GI_{50} values show that ANI-7 produces a GI_{50} value of 0.38 μM in MCF-7 cells, whereas values of 3.0-42 μM are observed in cell lines from lung, colon, ovary, neuronal, glial, prostate, and pancreas. The only other tumor type that shows appreciable growth inhibition by ANI-7 is the A431 vulva cell line (GI_{50} of 0.51μM)[1][1].

ANI-7 potently inhibits the growth of T47D, ZR-75-1, MCF-7, SKBR3, and MDA-MB-468 breast cancer cells (GI_{50} range of 0.16-0.38 μM), moderately inhibits the growth of BT20 and BT474 cells (GI50 range of 1-2 μM), and essentially fails to inhibit the growth of MDA-MB-231 and MCF10A cells (GI_{50} range of 17-26 μM). Moreover, ANI-7 maintained its ability to inhibit the growth of drug-resistant cells (MCF-7/VP16: GI_{50} of 0.21 μM)[1].

References:

[1]. ilbert J, et al. (Z)-2-(3,4-Dichlorophenyl)-3-(1H-Pyrrol-2-yl)Acrylonitrile Exhibits Selective Antitumor Activity in Breast Cancer Cell Lines via the Aryl Hydrocarbon Receptor Pathway. Mol Pharmacol. 2018 Feb;93(2):168-177.

[2]. Baker JR, et al. Dichlorophenylacrylonitriles as AhR Ligands That Display Selective Breast Cancer Cytotoxicity in vitro. ChemMedChem. 2018 Jul 18;13(14):1447-1458.

[3]. Mark Tarleton, et al. Library synthesis and cytotoxicity of a family of 2-phenylacrylonitriles and discovery of an estrogen dependent breast cancer lead compound. Medicinal Chemistry Communication. January 20112. (1):31-37.

CAIndexNames:

Benzeneacetonitrile, 3,4-dichloro-α-(1H-pyrrol-2-ylmethylene)-, (αZ)-

SMILES:

ClC1=CC(/C(C#N)=C/C2=CC=CN2)=CC=C1Cl

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