ANI-7

Cat. No.: HY-117102

CAS No.: 931417-26-4

Molecular Formula: $C_{13}H_8Cl_2N_2$

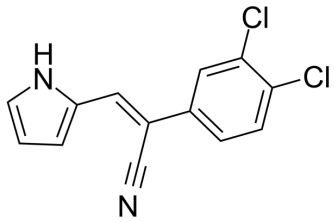
Molecular Weight: 263.12

Target: Aryl Hydrocarbon Receptor; Checkpoint Kinase (Chk)

Pathway: Immunology/Inflammation; Cell Cycle/DNA Damage

Storage:

Powder	-20°C	3 years
	4°C	2 years
In solvent	-80°C	2 years
	-20°C	1 year



SOLVENT & SOLUBILITY

In Vitro

DMSO : 20.83 mg/mL (79.17 mM; Need ultrasonic)

Preparing Stock Solutions	Solvent / Mass / Concentration	1 mg	5 mg	10 mg
	1 mM	3.8005 mL	19.0027 mL	38.0055 mL
	5 mM	0.7601 mL	3.8005 mL	7.6011 mL
	10 mM	0.3801 mL	1.9003 mL	3.8005 mL

Please refer to the solubility information to select the appropriate solvent.

In Vivo

1. Add each solvent one by one: 10% DMSO >> 40% PEG300 >> 5% Tween-80 >> 45% saline
   Solubility: ≥ 2.08 mg/mL (7.91 mM); Clear solution
2. Add each solvent one by one: 10% DMSO >> 90% corn oil
   Solubility: ≥ 2.08 mg/mL (7.91 mM); Clear solution

BIOLOGICAL ACTIVITY

Description

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].

IC_{50} & Target		
	Aryl Hydrocarbon Receptor	Chk2

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 H2AXγ 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].
MCE has not independently confirmed the accuracy of these methods. They are for reference only.

Cell Cycle Analysis[1]

Cell Line:	MCF10A and MDA-MB-468 cells
Concentration:	2.5 μM
Incubation Time:	24 hours
Result:	Induced significant S-phase and G2 + M-phase cell cycle arrest in MDA-MB-468 cells, and negligible effect in normal breast MCF10A cells.

Western Blot Analysis[1]

Cell Line:	MDA-MB-468 cells
Concentration:	2 μM
Incubation Time:	12 hours, 24 hours
Result:	Resulted in a significant increase in the content and phosphorylation of CHK2 (25-fold increase),and induced a significant increase in H2AXγ (3.5-fold increase).

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.

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

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