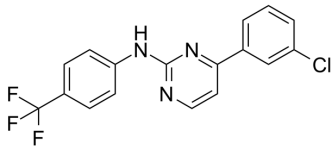
VAF347

Cat. No.:	HY-135750
CAS No.:	574759-62-9
Molecular Formula:	$C_{17}H_{11}ClF_3N_3$
Molecular Weight:	349.74
Target:	Aryl Hydrocarbon Receptor
Pathway:	Immunology/Inflammation
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 : 31.25 mg/mL (89.35 mM; Need ultrasonic)

Preparing Stock Solutions	Solvent / Mass / Concentration	1 mg	5 mg	10 mg
	1 mM	2.8593 mL	14.2963 mL	28.5927 mL
	5 mM	0.5719 mL	2.8593 mL	5.7185 mL
	10 mM	0.2859 mL	1.4296 mL	2.8593 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 (5.95 mM); Suspended solution; Need ultrasonic
2. Add each solvent one by one: 10% DMSO >> 90% corn oil
   Solubility: ≥ 2.08 mg/mL (5.95 mM); Clear solution

BIOLOGICAL ACTIVITY

Description

VAF347 is a cell permeable and highly affinity aryl hydrocarbon receptor (AhR) agonist and induces AhR signaling. VAF347 inhibits the development of $CD14^+CD11b^+$ monocytes from granulo-monocytic (GM stage) precursors. VAF347 has anti-inflammatory effects[1].

IC_{50} & Target

Aryl hydrocarbon receptor[1]

In Vitro

VAF347 (0.01-20 μM; 48-72 hours; HL-60 cells) treatment enhances retinoic acid-induced cell cycle arrest[1].
VAF347 (20 μM; 48 hours; HL-60 cells) treatment augments retinoic acid-induced expression of AhR, Lyn, Vav1, and c-Cbl as well as p47phox. Several interactions of partners in the signalsome appear to be enhanced: Fgr interaction with c-Cbl, CD38, and with pS259c-Raf and AhR interaction with c-Cbl and Lyn[1].

VAF347 inhibits IL-4$^+$ GM-CSF induced IL-6 production in MM1 cells with an IC_{50} of ~5 nM[2].

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

Cell Cycle Analysis[1]

Cell Line:	HL-60 cells
Concentration:	10 nM, 100 nM, 1 μM, 10 μM, 20 μM
Incubation Time:	48 hours or 72 hours
Result:	Enhanced retinoic acid-induced cell cycle arrest in G1/0.

Western Blot Analysis[1]

Cell Line:	HL-60 cells
Concentration:	20 μM
Incubation Time:	48 hours
Result:	Augmented retinoic acid-induced expression of AhR, Lyn, Vav1, and c-Cbl as well as p47phox.

In Vivo

In wild-type mice, VAF347 treatment leads to a strong reduction of total serum IgE levels compared with vehicle-treated animals. IL-5 levels in the bronchoalveolar fluid are inhibited to a comparable degree. AhR-deficient mice are resistant to the VAF347's ability to block allergic lung inflammation in vivo[2].

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

CUSTOMER VALIDATION

- J Nutr Biochem. 2023 Sep 2;109436.

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REFERENCES

[1]. Ibabao CN, et al. The AhR agonist VAF347 augments retinoic acid-induced differentiation in leukemia cells. FEBS Open Bio. 2015 Apr 8;5:308-18.

[2]. B Paige Lawrence, et al. Activation of the aryl hydrocarbon receptor is essential for mediating the anti-inflammatory effects of a novel low-molecular-weight compound. Blood. 2008 Aug 15;112(4):1158-65.

Tel: 609-228-6898 Fax: 609-228-5909 E-mail: tech@MedChemExpress.com

Address: 1 Deer Park Dr, Suite Q, Monmouth Junction, NJ 08852, USA