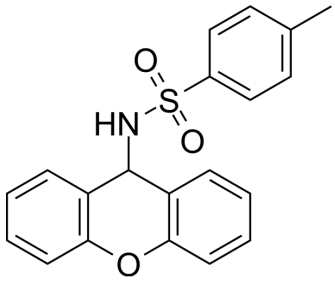
AH-7614

Cat. No.:	HY-19996
CAS No.:	6326-06-3
Molecular Formula:	$C_{20}H_{17}NO_3S$
Molecular Weight:	351.42
Target:	Free Fatty Acid Receptor
Pathway:	GPCR/G Protein
Storage:	Powder -20°C 3 years 4°C 2 years In solvent -80°C 6 months -20°C 1 month



SOLVENT & SOLUBILITY

In Vitro

DMSO : 100 mg/mL (284.56 mM; Need ultrasonic)

Preparing Stock Solutions	Solvent / Mass / Concentration	1 mg	5 mg	10 mg
	1 mM	2.8456 mL	14.2280 mL	28.4560 mL
	5 mM	0.5691 mL	2.8456 mL	5.6912 mL
	10 mM	0.2846 mL	1.4228 mL	2.8456 mL

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

In Vivo

1. Add each solvent one by one: 10% DMSO >> 90% corn oil
   Solubility: ≥ 2.5 mg/mL (7.11 mM); Clear solution

BIOLOGICAL ACTIVITY

Description

AH-7614 is a potent and selective FFA4 (GPR120) antagonist, with pIC_{50}s of 7.1, 8.1, and 8.1 for human, mouse, and rat FFA4, respectively. AH-7614 has selectivity for FFA4 over FFA1 (pIC_{50}<4.6). AH-7614 is able to block effects of both the polyunsaturated ω-6 fatty acid linoleic acid and the synthetic FFA4 agonist[1][2].

IC_{50} & Target

pIC50: 7.1 (human FFA4)[1]

In Vitro

AH-7614 (compound 39) (0.063-1 μM) blocks intracellular Ca^{2+} response induced by both linoleic acid and FFAR4 agonist in FFA4 expressing U2OS cells[1].
AH-7614 (100 μM) abolishes the enhancement in glucose-stimulated insulin secretion by GSK137647A in NCI-H716 cells[1].
AH-7614 (0.001-10 μM; 15 min) blocks TUG-891-mediated internalization of FFA4 from the cell surface (pIC_{50}=7.70)[2].
AH-7614 (10 μM; 30 min) blocks agonist-induced elevation of intracellular inositol monophosphates and phosphorylation of FFA4[2].

	MCE has not independently confirmed the accuracy of these methods. They are for reference only.
In Vivo	AH7614 (50 μg; intratumoral injection once every 4 d for 20 d) reduces the tumor growth in mice[3]. AH7614 (50 μg; intratumoral injection one day prior to epirubicin injection) enhances cancer cell sensitivity to the chemotherapy and inhibits tumor progression by blocking GPR120 signaling in combination with Epirubicin[3]. MCE has not independently confirmed the accuracy of these methods. They are for reference only.

REFERENCES

[1]. Sparks SM, et, al. Identification of diarylsulfonamides as agonists of the free fatty acid receptor 4 (FFA4/GPR120). Bioorg Med Chem Lett. 2014 Jul 15;24(14):3100-3.

[2]. Watterson KR, et, al. Probe-Dependent Negative Allosteric Modulators of the Long-Chain Free Fatty Acid Receptor FFA4. Mol Pharmacol. 2017 Jun;91(6):630-641.

[3]. Wang X, et, al. Fatty acid receptor GPR120 promotes breast cancer chemoresistance by upregulating ABC transporters expression and fatty acid synthesis. EBioMedicine. 2019 Feb;40:251-262.

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