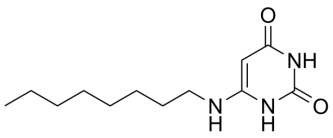
6-OAU

Cat. No.:	HY-12764
CAS No.:	83797-69-7
Molecular Formula:	$C_{12}H_{21}N_3O_2$
Molecular Weight:	239.31
Target:	GPR84; ERK; Bacterial; Antibiotic
Pathway:	GPCR/G Protein; MAPK/ERK Pathway; Stem Cell/Wnt; Anti-infection
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 : 25 mg/mL (104.47 mM; Need ultrasonic)

Preparing Stock Solutions	Solvent / Mass / Concentration	1 mg	5 mg	10 mg
	1 mM	4.1787 mL	20.8934 mL	41.7868 mL
	5 mM	0.8357 mL	4.1787 mL	8.3574 mL
	10 mM	0.4179 mL	2.0893 mL	4.1787 mL

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

BIOLOGICAL ACTIVITY

Description

6-OAU (GTPL5846) (6-n-octylaminouracil) is an GPR84 (G protein-coupled receptor 84) agonist, with an EC_{50} value of 105 nM. 6-OAU works as a chemoattractant to both PMNs and macrophages, and amplifies the proinflammatory cytokine IL-8, shows proinflammatory function. 6-OAU also displays anti-bacterial function[1][2].

IC_{50} & Target

ERK

In Vitro

GPR84 gene exhibits high expression in human polymorphonuclear leukocytes (PMNs) and macrophages, 6-OAU acts on proinflammatory function by activitating GPR84[1].

6-OAU (0.01 nM-0.1 mM; 1 h) activates human GPR84 in the presence of G_{qi5} chimera with an EC_{50} value of 105 nM in HEK293 cells[1].

6-OAU (0, 6.25, 200 μM; 30 min) stimulates [^{35}S]GTP binding, accumulates phosphoinositides, and induces GPR84-EGFP internalization in a GPR84-dependent manner[1].

6-OAU (1 nM-1 mM; 1 h) provokes chemotaxis of PMNs in a concentration-dependent manner with an EC_{50} value of 318 nM[1].

6-OAU (0-10 μM; 4 h) increases the secretion of IL-8 from LPS-stimulated PMNs[1].

6-OAU (0-0.4 μM; 16 h) also amplifies TNF-α production from U937 macrophages[1].
6-OAU (2 μM; 4 h) decreases ERK phosphorylation and MCP-1 protein expression, (2 μM; 48 h) decreases MCP-1 secretion in macrophages [2].
6-OAU (2 μM; 24 h) reduces ROS production during B. abortus infection in RAW264.7 cells[2].
6-OAU (2 μM; 0, 30, and 60 min) inhibits adhesion and Brucella uptake in RAW264.7 cells and (2 μM; 30 min) shows anti-infection against Brucella and Salmonella infection[2].
MCE has not independently confirmed the accuracy of these methods. They are for reference only.

Cell Viability Assay[2]

Cell Line:	B. abortus
Concentration:	0, 0.02, 0.2, 2 μM
Incubation Time:	0, 2, 24, 48, 72 hours
Result:	Decreased B. abortus survivability begin at 48 h with a dose of 2 μM.

Western Blot Analysis[2]

Cell Line:	RAW264.7 cells infected with B. abortus
Concentration:	2 μM
Incubation Time:	4 hours
Result:	Reduced ERK phosphorylation and MALT1 expression in RAW264.7 macrophages.

In Vivo

6-OAU activates GPR84 and results in making an inflammatory condition through chemokine production and chemotaxis in vivo[1].
6-OAU (10 mg/kg; i.v.) raises the blood CXCL1 level in rats[1].
6-OAU (1 mg/mL; s.c.) attracts both PMNs and macrophages into the air pouch[1].
6-OAU (2 μM, 100 mL/mouse; s.c.) augments resistance to Brucella infection, and reduces bacterial proliferation in spleens and livers[2].
MCE has not independently confirmed the accuracy of these methods. They are for reference only.

Animal Model:	Female Lewis rats(4-week-old)[1]
Dosage:	10 mg/kg
Administration:	Intravenous injection; collecting blood 3 h after injection
Result:	Increased the elevation of a chemokine, CXCL1 concentration in the serum peaking at 3 h after the injection.

Animal Model:	Rat air pouch model (4-week-old female rats)[1]
Dosage:	1 mg/mL (PBS)
Administration:	Subcutaneous injection; washing the cavity 4 h after injection
Result:	Attracted both PMNs and macrophages into the air pouch, peaking at 4 h after the injection.

Animal Model:	ICR female mice (7-week-old)[2]

Dosage:	2 μM (100 μL/mouse)
Administration:	Oral average; 7 days and another 14 days after treated mouse with B. abortus (2 × 105 CFU /100 μL; i.p.)
Result:	Reduced bacterial proliferation in the liver and spleen, and decreased IFN-γ but augmented IL-6 serum level. Lowed splenic weight of mice and splenic proliferation.

REFERENCES

[1]. Reyes AWB, et al. Immune-metabolic receptor GPR84 surrogate and endogenous agonists, 6-OAU and lauric acid, alter Brucella abortus 544 infection in both in vitro and in vivo systems. Microb Pathog. 2021 Sep. 158:105079.

[2]. Suzuki M, et al. Medium-chain fatty acid-sensing receptor, GPR84, is a proinflammatory receptor. J Biol Chem. 2013 Apr 12;288(15):10684-91.

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