BML-111

Cat. No.: HY-100450

CAS No.: 78606-80-1

Molecular Formula: $C_8H_{16}O_5$

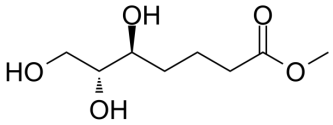
Molecular Weight: 192.21

Target: Angiotensin-converting Enzyme (ACE)

Pathway: Metabolic Enzyme/Protease

Storage:

Pure form	-20°C	3 years
In solvent	-80°C	6 months
	-20°C	1 month



SOLVENT & SOLUBILITY

In Vitro

DMSO : 100 mg/mL (520.26 mM; Need ultrasonic)

Preparing Stock Solutions	Mass / Solvent / Concentration	1 mg	5 mg	10 mg
	1 mM	5.2026 mL	26.0132 mL	52.0264 mL
	5 mM	1.0405 mL	5.2026 mL	10.4053 mL
	10 mM	0.5203 mL	2.6013 mL	5.2026 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: ≥ 2.5 mg/mL (13.01 mM); Clear solution
2. Add each solvent one by one: 10% DMSO >> 90% (20% SBE-β-CD in saline)
   Solubility: ≥ 2.5 mg/mL (13.01 mM); Clear solution
3. Add each solvent one by one: 10% DMSO >> 90% corn oil
   Solubility: ≥ 2.5 mg/mL (13.01 mM); Clear solution

BIOLOGICAL ACTIVITY

Description

BML-111, a lipoxin A_4 analog, is a lipoxin A_4 receptor agonist. BML-111 represses the activity of angiotensin converting enzyme (ACE) and increases the activity of angiotensinconverting enzyme 2 (ACE2). BML-111 has antiangiogenic, antitumor and anti-inflammatory properties[1][2].

IC_{50} & Target

Lipoxin A4 receptor[1]
Angiotensin converting enzyme (ACE)[2]

In Vitro

In H22 cells, BML-111 inhibits the production of vascular endothelial growth factor and reduces hypoxia-inducible factor-1α

level[1].
BML-111 inhibits leukotriene B4-induced cellular migration with an IC_{50} of 5 nM[3].
MCE has not independently confirmed the accuracy of these methods. They are for reference only.

In Vivo

BML-111 (1 mg/kg; intraperitoneal injection; for 15 days; male Imprinting Control Region mice) treatment suppresses tumor-related angiogenesis and tumor growth in vivo. BML-111 also enhances the in situ apoptosis while inhibiting macrophage infiltration in tumor tissue[1].
BML-111 protects LPS-induced acute lung injury and LPS/D-GalN-induced acute liver injury. BML-111 represses the activity of ACE, but increases the activity of ACE2. BML-111 decreases the expression levels of ACE, AngII, and AngII type 1 receptor (AT1R), meanwhile increases the levels of ACE2, angiotensin-(1-7) (Ang-1-7), and Mas[2].
MCE has not independently confirmed the accuracy of these methods. They are for reference only.

Animal Model:	Male Imprinting Control Region mice (5-6-week-old,18-22 g) injected with H22 cells[1]
Dosage:	1 mg/kg
Administration:	Intraperitoneal injection; injected 5 minutes before and 4 hours after H22 cell inoculation, then every 12 hours for 2 consecutive days, then daily in an additional 3 days and every other day for the last 10 days
Result:	Suppressed tumor-related angiogenesis and tumor growth in vivo.

REFERENCES

[1]. Ying Chen, et al. Lipoxin A4 and Its Analogue Suppress the Tumor Growth of Transplanted H22 in Mice: The Role of Antiangiogenesis. Mol Cancer Ther. 2010 Aug;9(8):2164-74.

[2]. Qiong-Feng Chen, et al. BML-111, a Lipoxin Receptor Agonist, Protects Against Acute Injury via Regulating the Renin Angiotensin-Aldosterone System. Prostaglandins Other Lipid Mediat. 2019 Feb;140:9-17.

[3]. T H Lee, et al. Inhibition of Leukotriene B4-induced Neutrophil Migration by Lipoxin A4: Structure-Function Relationships. Biochem Biophys Res Commun. 1991 Nov 14;180(3):1416-21.

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