# BML-111

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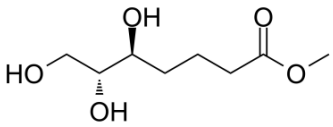
| **Cat. No.:** | HY-100450 |
| **CAS No.:** | 78606-80-1 |
| **Molecular Formula:** | $C_8H_{16}O_5$ |
| **Molecular Weight:** | 192.21 |
| **Target:** | Angiotensin-converting Enzyme (ACE) |
| **Pathway:** | Metabolic Enzyme/Protease |
| **Storage:** | Please store the product under the recommended conditions in the COA. |




## BIOLOGICAL ACTIVITY

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| **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]<br>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].<br>BML-111 inhibits leukotriene B4-induced cellular migration with an $IC_{50}$ of 5 nM[3]. |
| **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].<br>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]. |

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| **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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