

Lysophosphatidylcholines

Cat. No.:	HY-139414									
CAS No.:	9008-30-4									
Target:	Interleukin Related; p38 MAPK; ERK; Apoptosis									
Pathway:	Immunology/Inflammation; MAPK/ERK Pathway; Stem Cell/Wnt; Apoptosis									
Storage:	<table border="0"> <tr> <td>Powder</td> <td>-20°C</td> <td>3 years</td> </tr> <tr> <td>In solvent</td> <td>-80°C</td> <td>6 months</td> </tr> <tr> <td></td> <td>-20°C</td> <td>1 month</td> </tr> </table>	Powder	-20°C	3 years	In solvent	-80°C	6 months		-20°C	1 month
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Lysophosphatidylcholines

SOLVENT & SOLUBILITY

In Vitro	<p>Methanol : 25 mg/mL (Need ultrasonic)</p> <p>DMSO : < 1 mg/mL (ultrasonic;warming;heat to 60°C) (insoluble or slightly soluble)</p> <p>H₂O : < 0.1 mg/mL (ultrasonic) (insoluble)</p>
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BIOLOGICAL ACTIVITY

Description	<p>Lysophosphatidylcholines is an orally active lysolipid and a component of oxidized low density lipoprotein (LDL). Lysophosphatidylcholines induces cell injury, the production of IL-1β and apoptosis. Lysophosphatidylcholines has a proactive effect on sepsis^{[1][2][3][4]}.</p>
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IC₅₀ & Target	IL-1 β	ERK1	ERK2
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In Vitro	<p>Lysophosphatidylcholine (3-100 μM, 24 h) reduced HUVEC viability^[1].</p> <p>Lysophosphatidylcholines (12.5μM, 4 h) upregulates gene expression of IL-1β in human peripheral blood monocytes^[2].</p> <p>Lysophosphatidylcholines (75μM, 24 h) induces apoptosis in HUVEC through a p38-mitogen-activated protein kinase-dependent mechanism^[3].</p> <p>MCE has not independently confirmed the accuracy of these methods. They are for reference only.</p>
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Cell Cytotoxicity Assay^[1]

Cell Line:	HUVEC
Concentration:	3 μ M, 10 μ M, 30 μ M, 100 μ M
Incubation Time:	24 h
Result:	Reduced HUVEC viability in a concentration-dependent manner.

Western Blot Analysis^[3]

Cell Line:	HUVEC
Concentration:	75 μ M

	Incubation Time:	24 h
	Result:	Showed both ERK1/2 and p38-MAPK phosphorylation.
In Vivo	Lysophosphatidylcholines (0.1-20 mg/kg, Oral, single dose) protects against sepsis-induced lethality on albino ICR mice ^[4] . Lysophosphatidylcholines (0.1-20 mg/kg, Oral, single dose) enhances bacterial clearance, blocks cecal ligation and puncture (CLP)-induced neutrophil deactivation and increases bactericidal activity of neutrophils ^[4] . MCE has not independently confirmed the accuracy of these methods. They are for reference only.	
	Animal Model:	Albino ICR mice ^[4]
	Dosage:	0.1 mg/kg, 1 mg/kg, 10 mg/kg, 20 mg/kg
	Administration:	Oral
	Result:	Provided significant protection against cecal ligation and puncture (CLP)-induced lethality at a dose of 1 mg/kg.

REFERENCES

- [1]. Kim E A, et al. Lysophosphatidylcholine induces endothelial cell injury by nitric oxide production through oxidative stress [J]. The Journal of Maternal-Fetal & Neonatal Medicine, 2009, 22(4): 325-331.
- [2]. Liu-Wu Y, et al. Lysophosphatidylcholine induces the production of IL-1 β by human monocytes [J]. Atherosclerosis, 1998, 137(2): 351-357.
- [3]. Takahashi M, et al. Lysophosphatidylcholine induces apoptosis in human endothelial cells through a p38-mitogen-activated protein kinase-dependent mechanism [J]. Atherosclerosis, 2002, 161(2): 387-394.
- [4]. Yan J J, et al. Therapeutic effects of lysophosphatidylcholine in experimental sepsis [J]. Nature medicine, 2004, 10(2): 161-167.

Caution: Product has not been fully validated for medical applications. For research use only.

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