Lipopolysaccharides, from E. coli O55:B5

Cat. No.: HY-D1056

Target: Toll-like Receptor (TLR)

Pathway: Immunology/Inflammation

Storage:

Powder	-20°C	3 years
	4°C	2 years
In solvent	-80°C	6 months
	-20°C	1 month

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SOLVENT & SOLUBILITY

In Vitro	DMSO : 14.29 mg/mL (ultrasonic and warming and heat to 60°C) H_2O : 5 mg/mL (Need ultrasonic)
In Vivo	1. Add each solvent one by one: PBS Solubility: 8.33 mg/mL (Infinity mM); Clear solution; Need ultrasonic and warming and heat to 60°C 2. Add each solvent one by one: 10% DMSO >> 90% (20% SBE-β-CD in saline) Solubility: ≥ 1.43 mg/mL (Infinity mM); Clear solution

BIOLOGICAL ACTIVITY

Description	Lipopolysaccharides (LPS) is an endotoxin derived from the outer leaflet of the outer membrane of Gram-negative bacteria. Lipopolysaccharides consists of an antigen O-specific chain, a core oligosaccharide and lipid A. Lipopolysaccharides is a pathogenic associated molecular pattern (PAMP) that activates the immune system. Lipopolysaccharides activates TLR-4 on immune cells[1][2][3]. This product is derived from Escherichia coli O55:B5. Lipopolysaccharides induces secretion of cell migrasome[4].
IC_{50} & Target	TLR4
In Vitro	Lipopolysaccharides (10–80 μg/mL) selectively decreases THir (tyrosine hydroxylase immunoreactive) cells and increases culture media levels of interleukin1β (IL-1β) and tumor necrosis factor-α (TNF-α) as well as nitrite (an index of nitric oxide (NO) production)[5]. MCE has not independently confirmed the accuracy of these methods. They are for reference only.
In Vivo	Lipopolysaccharides can be used in animal modeling to construct a mouse liver inflammation model. Lipopolysaccharides (1.5 mg/kg; i.p.; once) induces sickness and hypothermia in mice, and induces a greater and more prolonged sickness response in adult male mice[3]. MCE has not independently confirmed the accuracy of these methods. They are for reference only.

Animal Model:	Female and male CD1 mice[3]
Dosage:	1.5mg/kg
Administration:	Intraperitoneal injection, once
Result:	Induced sickness behavior in all mice, but adult mice displayed more sickness than pubertal mice and adult males remained sick for a longer period of time than adult females. Caused a decrease in body temperature for all mice, but this decrease was greatest in adult males. Increased pro- and anti-inflammatory cytokines at various levels in pubertal and adult male and female mice, resulted in age and sex differences in cytokine concentrations following immune challenge. Only adult males and females treated with LPS displayed significantly more IL-6 than their saline controls, and pubertal males and females and adult females displayed significantly more IL-10 than their saline controls. All the mice displayed significantly more IL-12 and TNF-α than their saline controls.

CUSTOMER VALIDATION

- Cell Res. 2023 Jul 17.
- Immunity. 2024 Feb 16:S1074-7613(24)00044-X.
- Adv Mater. 2024 Feb 1:e2311964.
- Adv Mater. 2023 Jan 19;e2210787.
- Adv Funct Mater. 10 March 2022.

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REFERENCES

[1]. Cai KC, et al. Age and sex differences in immune response following LPS treatment in mice. Brain Behav Immun. 2016 Nov;58:327-337.

[2]. Gayle DA, et al. Lipopolysaccharide (LPS)-induced dopamine cell loss in culture: roles of tumor necrosis factor-alpha, interleukin-1beta, and nitric oxide. Brain Res Dev Brain Res. 2002 Jan 31;133(1):27-35.

[3]. Ying Liu, et al. Podocyte-Released Migrasomes in Urine Serve as an Indicator for Early Podocyte Injury. Kidney Dis (Basel). 2020 Nov;6(6):422-433.

[4]. Kabanov DS, et al. Structural analysis of lipopolysaccharides from Gram-negative bacteria. Biochemistry (Mosc). 2010 Apr;75(4):383-404.

[5]. Heinrichs DE, et al. Molecular basis for structural diversity in the core regions of the lipopolysaccharides of Escherichia coli and Salmonella enterica. Mol Microbiol. 1998 Oct;30(2):221-32.

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

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