Lipopolysaccharide from E. coli (O55:B5)

Lipopolysaccharide extracted from *E. coli* (wild-type S-form, serotype O55:B5)

Catalog #100-1270 1 mL 1 mg/mL

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Product Description

Lipopolysaccharide from *E. coli* (O55:B5) is a lipopolysaccharide (LPS) derived from the O55:B5 serotype of the Gram-negative bacteria, *Escherichia coli*. LPS are glycolipid constituents that reside on the outer membranes of Gram-negative bacteria (Kitchens RL et al.). LPS are composed of a lipid A, a core oligosaccharide, and an O antigen. LPS protects bacteria against bile salts and lipophilic antibiotics by maintaining the outer integrity of the cell membrane (Bäckhed F et al.). Lipopolysaccharides from *E. coli* (O55:B5) is predominantly recognized by toll-like receptor 4 (TLR4), which leads to the activation of NF-κβ, a protein complex which plays a key role in regulating immune response (Kuzmich N et al.). Activation of NF-κβ can trigger increased production of pro-inflammatory cytokines IL-1 and TNF-α by macrophages (Matuschak GM et al.). This LPS can also interact with CD14 to activate phospholipase Cγ2 and kinases of the Src family, trigger influxes of extracellular Ca^{2+}, as well as calcineurin-dependent translocation of the nuclear factor of activated T cells (NFAT) family of transcription factors (Li CC et al.).

When added to ImmunoCult™-SF Macrophage Medium (Catalog #10961), stimulation with Lipopolysaccharide from *E. coli* (O55:B5) and IFN-γ supports the polarization to M1 (classically activated) macrophages.

WARNING: This product is highly pyrogenic. Avoid all means by which the product may enter the bloodstream. Please refer to the Safety Data Sheet (SDS) for hazard information.

Product Information

Formulation:	1 mg/mL stabilized in sterile, double-distilled water (ddH_2O), without any additives.
Source:	*E. coli* (wild-type S-form, serotype O55:B5)

Specifications

Activity:	0.01 - 1 µg/mL in vitro and 5 - 15 mg/kg in vivo in animal rodent models. Optimal concentration is dependent upon cell type, species, and application. Does not activate any TLR other than TLR4 as tested up to 1 µg/mL in relevant cellular systems (see Figure 1).
Purity:	Ultrapure (≥ 99.9%). No detectable DNA, RNA, or protein traces.
Endotoxin Content:	Measured by kinetic turbidimetric Limulus amebocyte lysate (LAL) and is > 5 x 10^6 EU/mL protein.

Preparation and Storage

Storage:	Store at 2 - 8°C.
Stability:	Stable as supplied for 2 years from date of receipt. Diluted solutions are stable for 12 hours at 2 - 8°C.
Preparation:	Warm to room temperature (15 - 25°C) and dilute as necessary in phosphate-buffered saline (PBS; e.g. Catalog #37350), endotoxin-free ddH_2O, or 0.9% NaCl solution before use. For example, to prepare a 100 µg/mL solution in PBS, add 100 µL of LPS to 900 µL of PBS. Use the diluted solution immediately; do not store.

Data

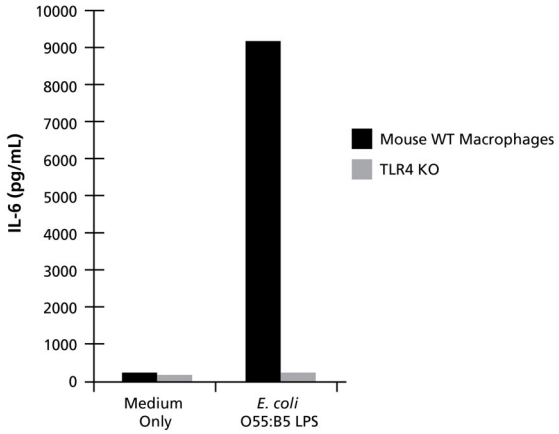

Figure 1. Stimulation with Lipopolysaccharide from *E. coli* (O55:B5) Resulted in High Production of IL-6 through TLR4-specific Activation

The biological activity of Lipopolysaccharide (LPS) from *E. coli* (O55:B5) was assessed by its ability to activate TLR4 in both wild-type and TLR4 knockout (KO) mouse macrophages, with a medium-only negative control. When stimulated with 1 µg/mL of LPS from *E. coli* (O55:B5), wild-type mouse macrophages produced ≥ 9200 pg/mL of IL-6, as measured by ELISA in cell culture supernatants after 24 hours. IL-6 production in TLR4 KO macrophages was comparable to the medium-only negative control, indicating that activation by LPS from *E. coli* (O55:B5) did not activate any TLR other than TLR4. The optimal concentration required for activation is 0.01 - 1 µg/mL.

References

Bäckhed F et al. (2003) Structural requirements for TLR4-mediated LPS signalling: a biological role for LPS modifications. Microbes Infect 5(12): 1057–63.

Kitchens RL & Munford RS. (1995) Enzymatically deacylated lipopolysaccharide (LPS) can antagonize LPS at multiple sites in the LPS recognition pathway. J Biol Chem 270(17): 9904–10.

Kuzmich NN et al. (2017) TLR4 signaling pathway modulators as potential therapeutics in inflammation and sepsis. Vaccines 5(4): 34.

Li CC et al. (2015) Suppression of dendritic cell-derived IL-12 by endogenous glucocorticoids is protective in LPS-induced sepsis. PLOS Biol 13(10): e1002269.

Matuschak GM et al. (2010) Acute hypoxia decreases E. coli LPS-induced cytokine production and NF-κB activation in alveolar macrophages. Respir Physiol Neurobiol 172(1–2): 63–71.