Product Data Sheet

Product Name:	Lipopolysaccharides from Escherichia coli O111:B4
Cat. No.:	GC19203

Chemical Properties

Cas No.			
分子式		分子量	
溶解度	Soluble in water (5 mg/ml) or cell culture medium (1 mg/ml)	储存条件	Store at 2-8°C
General tips	For obtaining a higher solubility , please warm the tube at 37 ℃ and shake it in the ultrasonic bath for a while.Stock solution can be stored below -20℃ for several months.		
Shipping Condition	Evaluation sample solution : ship with blue ice All other available size: ship with RT , or blue ice upon request.		

Structure

Protocol

Cell experiment [1]:

Cell lines	Human cancer cell line HT-29
Preparation Method	HT-29 cells were incubated at 37℃ in a humidified atmosphere of 5% CO2 in low-D-glucose (16.67 mM) McCoy's 5a Medium Modified supplemented with 10% v/v heat-inactivated FBS, 2 mM L-glutamine, and 1% penicillin/streptomycin.
Reaction Conditions	Prior to any treatment, cells were allowed to reach confluence in plate wells, and then monolayers were exposed to a range of concentrations of carrageenans (10, 50, and 100 μg x mL^{-1}, final value), lipopolysaccharides (10 μg x mL^{-1}, final value). Furthermore, stress model was induced by ethanol (10%).
Applications	Mixtures of lipopolysaccharides and carrageenans exhibited a tendency toward the reference profile not exposed to ethanol, but at a rate less rapid than that of cells preincubated with the carrageenan alone. In the presence of lipopolysaccharides, κ/β-carrageenan remained active, whereas the other carrageenans had no activity. The differences.

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Animal experiment [2]:

Animal models	Male Sprague-Dawley rats (200 – 250 g)
Preparation Method	Animals were housed with free access to food and water. Lipopolysaccharide from Salmonella thyphosa (Sigma) dissolved in endotoxin-free saline was used for intraperitoneal injection. Animals were sacrificed after 2, 6, 12, and 24 h, and pancreas, liver, kidney, lung, brain, and intestine were processed.
Dosage form	30 mg/kg
Applications	Lipopolysaccharide treatment could induce p8 mRNA expression in the pancreas. Maximal induction (31fold) was observed after 12 h and expression remained significantly elevated after 24 h. p8 mRNA was also overexpressed after Lipopolysaccharide intraperitoneal injection in liver and kidney. Maximal p8 mRNA expression was obtained after 6 and 12 h of the LPS treatment in kidney and liver respectively. Induction was of 10 and 8fold in liver and kidney respectively.

References:
[1]. Sokolova EV, et al. Effect of carrageenans alone and in combination with casein or lipopolysaccharide on human epithelial intestinal HT-29 cells. J Biomed Mater Res A. 2017 Oct;105(10):2843-2850.
[2]. Jiang YF, et al. Lipopolysaccharides induce p8 mRNA expression in vivo and in vitro. Biochem Biophys Res Commun. 1999 Jul 14;260(3):686-90.

Background

This product is extracted from E. coli serotype O111:B4 and purified by gel filtration. The source strain is from a private collection. This LPS serotype has been used to stimulate B-cells and induce NOS in human hepatocytes.

Lipopolysaccharides (LPSs) are characteristic components of the cell wall of Gram-negative bacteria. LPS and its lipid A moiety stimulate cells of the innate immune system by the Toll-like receptor 4 (TLR4), a member of the Toll-like receptor protein family, which recognizes common pathogen-associated molecular-patterns (PAMPs).

Lipopolysaccharide (LPS) is vital to both the structural and functional integrity of the Gram-negative bacterial outer membrane. Ubiquitously expressed by all Gram-negative bacteria, and containing several well-conserved domains, lipopolysaccharide also serves as one of the primary targets of the innate arm of the mammalian immune system. The lipopolysaccharides have a profound effect on the mammalian immune system and are of great significance in the pathophysiology of many disease processes.[1]

In vitro study indicated that the bone resorption and the inhibition of collagen synthesis caused by lipopolysaccharide could be prevented by PB effectively. Lipopolysaccharide at a concentration of 10μg /ml inhibited bone collagen synthesis by 43% and PB reversed this inhibition in a dose-dependent manner. Even at concentrations as low as 5 μg/ml (PB: LPS =1:2) it reduced the bone-resorbing activity of the lipopolysaccharide by 85%. This effect was specific for resorption stimulated by lipopolysaccharide.[2]

Lipopolysaccharide preconditioning to mice obviously reduced coelenterazine-Induced fluorescent lesions of Colon26 cells at 7 and 14 days after the intraportal inoculation of Colon26 cells, which expressed Nano-lantern, in comparison to control mice. Moreover, lipopolysaccharide preconditioning significantly reduced the fluorescence intensity of tumors than that of the control mice at both 7 and 14 days after tumor inoculation as well as reduced the liver weight in comparison to control mice at 14 days. Results showed that tumor metastasis was exclusively found in the lungs but not liver. Lipopolysaccharide preconditioning also tended to reduce lung metastasis in vivo.[3]

References:
[1]. Erridge C, et al. Structure and function of lipopolysaccharides. Microbes Infect. 2002 Jul;4(8):837-51.
[2]. Harvey W, et al. In vitro inhibition of lipopolysaccharide-induced bone resorption by polymyxin B. Br J Exp Pathol. 1986 Oct;67(5):699-705.

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[3]. Nishikawa M, et al. Lipopolysaccharide preconditioning reduces liver metastasis of Colon26 cells by enhancing antitumor activity of natural killer cells and natural killer T cells in murine liver. J Gastroenterol Hepatol. 2021 Jul;36(7):1889-1898.

该产品从大肠杆菌血清型 O111:B4 中提取，并通过凝胶过滤进行纯化。源菌株来自私人收藏。这种 LPS 血清型已被用于刺激 B 细胞并在人肝细胞中诱导 NOS。

脂多糖 (LPS) 是革兰氏阴性菌细胞壁的特征成分。 LPS 及其脂质 A 部分通过 Toll 样受体 4 (TLR4) 刺激先天免疫系统的细胞，TLR4 是 Toll 样受体蛋白家族的一员，可识别常见的病原体相关分子模式 (PAMP)。\n

脂多糖 (LPS) 对于革兰氏阴性细菌外膜的结构和功能完整性至关重要。脂多糖由所有革兰氏阴性菌普遍表达，并包含几个保存完好的结构域，也是哺乳动物免疫系统先天性臂的主要靶标之一。脂多糖对哺乳动物的免疫系统具有深远的影响，在许多疾病过程的病理生理学中具有重要意义。[1]

体外研究表明，PB可有效阻止脂多糖引起的骨吸收和胶原合成抑制。浓度为 10μg/ml 的脂多糖抑制骨胶原合成达 43%，PB 以剂量依赖的方式逆转这种抑制作用。即使浓度低至 5 μg/ml（PB: LPS =1:2），它也会将脂多糖的骨吸收活性降低 85%。这种效应对脂多糖刺激的再吸收具有特异性。[2]

与对照小鼠相比，在门静脉内接种表达 Nano-lantern 的 Colon26 细胞后 7 天和 14 天，对小鼠进行脂多糖预处理明显减少了腔肠素诱导的 Colon26 细胞荧光损伤。此外，在肿瘤接种后 7 天和 14 天，脂多糖预处理显着降低了肿瘤的荧光强度，与对照小鼠相比，在 14 天时，与对照小鼠相比，肝脏重量也有所减轻。结果表明，肿瘤转移仅发生在肺部，而不是肝脏。脂多糖预处理也倾向于减少体内肺转移。[3]

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