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Data Article

Early LPS-induced ERK activation in retinal pigment epithelium cells is dependent on PIP₂-PLC[☆]



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Lipopolysaccharide (LPS)

ABSTRACT

This article presents additional data regarding the study “The phospholipase D pathway mediates the inflammatory response of the retinal pigment epithelium” [1]. The new data presented here show that short exposure of RPE cells to lipopolysaccharide (LPS) induces an early and transient activation of the extracellular signal-regulated kinase (ERK1/2). This early ERK1/2 activation is dependent on phosphatidylinositol bisphosphate-phospholipase C (PIP₂-PLC). On the contrary, neither the phospholipase D 1 (PLD1) nor the PLD2 inhibition is able to modulate the early ERK1/2 activation induced by LPS in RPE cells.

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Abbreviations: ERK, extracellular signal-regulated kinase; HRP, horseradish peroxidase; LPS, lipopolysaccharide; PIP₂-PLC, phosphatidylinositol bisphosphate-phospholipase C; PLD, phospholipase D; RPE, retinal pigment epithelium

[☆]The data presented here is additional data related to the publication “The phospholipase D pathway mediates the inflammatory response of the retinal pigment epithelium” Melina V. Mateos, Constanza B. Kamerbeek, Norma M. Giusto, Gabriela A. Salvador, *Int. J. Biochem. Cell. Biol.*, vol. 55, October 2014, pp. 119–128, doi: 10.1016/j.biocel.2014.08.016, Epub 2014 August 27.

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Specifications table

Subject area	Biochemistry
More specific sub- ject area	Cell biology
Type of data	WB images, bar graphs
How data was acquired	Western blot. Densitometry values were obtained using the ImageJ software
Data format	Raw and analyzed
Experimental factors	ARPE-19 cells were exposed to LPS. Pharmacological inhibitors of PLD1, PLD2 and PIP ₂ -PLC were used.
Experimental features	ERK1/2 activation was evaluated by Western blot
Data source location	Instituto de Investigaciones Bioquímicas de Bahía Blanca (INIBIBB), Universidad Nacional del Sur (UNS) and Consejo Nacional de Investigaciones Científicas y Técnicas (CONICET), 8000 Bahía Blanca, Argentina.
Data accessibility	Data is provided within the article

Value of the data

- The data can be useful to other scientists investigating the effects of LPS on RPE cells.
- The data provide additional information regarding the LPS-induced ERK1/2 signaling in RPE cells.
- Results shown here demonstrate that the early and the late LPS-induced ERK1/2 activation are differentially modulated by PIP2-PLC and PLD pathways.

1. Data

The data presented here show that in RPE cells, ERK1/2 activation induced by 5 min treatment with LPS depends on PIP₂-PLC but is not affected by classical PLDs inhibition.

2. Experimental design, materials and methods

2.1. Retinal-pigmented epithelium cell culture and treatments

Human retinal-pigmented epithelium cells (ARPE-19) were maintained in Dulbecco's Modified Eagle's Medium (DMEM) supplemented with 10% fetal bovine serum (FBS, Natocor, Argentina), 100 U/ml penicillin, 100 µg/ml streptomycin and 0.25 µg/ml amphotericin B at 37 °C under 5% CO₂. Confluent 35 mm diameter cell dishes were serum-starved for 2 h prior to stimulation for 5 min or 2 h with 10 µg/ml of *Pseudomonas aeruginosa* LPS in serum-free DMEM. Sterile ultra pure water was added to the control condition. In order to inhibit PIP₂-PLC and PLDs pathways ARPE-19 cells were preincubated with selective inhibitors for 1 h at 37 °C prior to cell stimulation with LPS. 0.15 µM EVJ was used to inhibit PLD1 activity and 0.5 µM APV to inhibit PLD2. For PIP₂-PLC inhibition, cells were preincubated with U73122 (10 µM). DMSO (vehicle of the inhibitors) was added to all conditions to achieve a final concentration of 0.025% [1,2].

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