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Lipoxin A4 (LXA4) attenuates oxidative stress-mediated response through TLR4 modulation in Primary Bronchial Epithelial Cells (PBECs)

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European Respiratory Journal 2024 64(suppl 68): PA3506; DOI: https://doi.org/10.1183/13993003.congress-2024.PA3506

This article appears in:

European Respiratory Journal

Vol 64 Issue suppl 68

Abstract

Innate immune system has an active functional role in the inflammatory response, oxidative stress-mediated mechanisms and resolution process. The resolution of inflammation is a dynamic process with highly regulated cellular and biochemical events. LXA4, the lead member of pro-resolving mediators, has anti-inflammatory properties, regulates immune response and attenuates oxidative stress.

The aim of our study was to investigate the effects of LXA4 on the expression and modulation of Toll-Like Receptors (TLRs) induced by oxidative stress in PBECs exposed to Cigarette Smoke Extract (CSE).

PBECs were cultured in presence or absence of 5% CSE, LXA4 (100nM) and FPR2/ALXR (Phormyl peptide Receptor 2–lipoxin receptor) functional blocking peptide, alone or in combination. TLR2 and TLR4 expression, LPS binding (ALEXA fluor LPS), and ROS production were evaluated in PBECs using flow cytometry analyses. IL-8 levels were evaluated in supernatants of PBECs.

We found that LXA4 significantly reduces TLR4 expression, LPS binding, ROS generation, and IL-8 release, induced by CSE treatment in PBECs. No differences were detected in TLR2 expression in the different experimental conditions. PBECs pretreatment with the FPR2/ALXR blocking peptide counteracted the effect of LXA4 on TLR4 expression, LPS binding, ROS and IL-8.

Our results show that LXA4 provides a protective role in attenuating inflammatory and oxidative effects induced by cigarette smoke in airway epithelium. We suggest a potential LXA4 involvement in the regulation of innate immunity system and susceptibility to inflammatory and infectious processes through modulation of TLR4 expression under oxidative stress conditions.

Article Info & Metrics Focus Previous Next

Article Information

vol. 64 no. suppl 68 Page: PA3506

DOI https://doi.org/10.1183/13993003.congress-2024.PA3506

Print ISSN 0903-1936

Online ISSN 1399-3003

History Published online 30 October 2024

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