Neutrophil Infiltration Is Partially Inhibited By EC-18 in the LPS-Induced Acute Lung Injury

Su-Hyun Shin, Ha-Reum Lee, Nina Yoo, Sun Young Yoon, Myung-Hwan Kim, Cheolwon Suh, Ki-Young Sohn, Heung-Jae Kim, Yong-Hae Han, Saeho Chong, and Jae Wha Kim

Blood 2015 126:2212;

Abstract

Acute lung injury (ALI) is an acute respiratory failure and linked closely to neutrophil accumulation. It can lead to acute respiratory distress syndrome (ARDS). Mouse model of ALI was established by lipopolysaccharide (LPS) administration. LPS, an outer membrane of gram negative bacteria, is considered as immune stimulator via recognition as pathogen-associated molecular patterns (PAMP). ALI and neutrophil infiltration were readily induced by intranasal injection of LPS. In this study, we investigated whether EC-18 treatment attenuates LPS-induced ALI. EC-18 (1-palmitoyl-2-linoleoyl-3-acetyl-rac-glycerol) is an immune modulator in the allergic asthma response through modulation of the balance between Th1 and Th2. To analyze the role of EC-18 in LPS-induced ALI mice, Balb/c mice were divided into three separate groups: control, LPS treated, and EC-18/LPS co-treated (n=5 per group). 25 mg/kg of LPS was administered by an intranasal route, and 250 mg/kg of EC-18 was orally administered. Mice were sacrificed after 15hrs, and various samples were collected. Bone marrow cells, whole blood cells, cells in lung and bronchoalveolar lavage fluid (BALF) were analyzed using complete blood count (CBC) assay. As results, the number of neutrophil in the bone marrow was decreased in the LPS treated group, and the circulating neutrophil, neutrophil in the lung and BALF were significantly increased in the LPS treated group. Neutrophils in the lung and BALF were dramatically decreased in the EC-18/LPS co-treated group. Also, Evans Blue staining of the lung indicated that capillary permeability was enhanced in the LPS

injected mice, and this permeability was lessened in the EC-18/LPS co-treated group as much as that of control. These findings suggested that EC-18 could effectively block neutrophil transmigration into the lung and vesicular leakage. Consequently, EC-18 could be utilized as a potential therapeutic agent for acute and chronic inflammation related disease like ALI.

Disclosures No relevant conflicts of interest to declare.

- * Asterisk with author names denotes non-ASH members.

Back to top

Previous

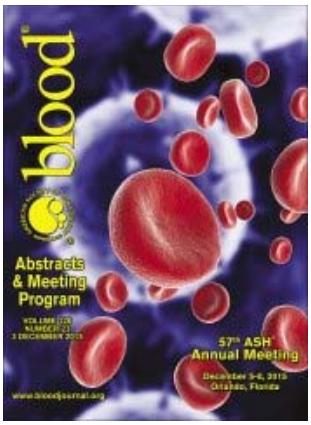

Table of Contents
Volume: 126
Issue: 23
Pages: 2212
DOI: https://doi.org/

Email
Alerts
Citation Tools
Request Permissions
Share

Cite This

Jump to

Current Issue
First Edition
Collections
All Issues
Abstracts
Video Library
About *Blood*
Subscriptions
Newsroom
Copyright
Public Access
Permissions
Submit to *Blood*
Blood App
Alerts
RSS
Contact Us
Order Reprints
Information for:
Authors
Subscribers
Institutions/Librarians
Advertising in *Blood*
Terms and Conditions

Blood Journals

Privacy Policy | Terms of Service | Contact Us