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REVIEWS

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Quercetin for inhibition of inflammatory responses and oxidative stress in lung injury model: a systematic review and meta-analysis

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Abstract

Acute lung injury (ALI) is a life-threatening clinical syndrome with high morbidity and mortality. The main pathological features of ALI are increased alveolar-capillary membrane permeability, edema, uncontrolled migration of neutrophils to the lungs, and diffuse alveolar damage, resulting in acute hypoxemic respiratory failure. We performed a systematic review and meta-analysis to elucidate the antioxidant activities of flavanols in a rat model of acute lung injury (ALI). PubMed, EMBASE, Scopus, ProQuest, Web of Science, and Google Scholar databases were searched to obtain the relevant papers. Nine studies with 343 rat models of ALI were included in this study. We investigated oxidative stress with the corresponding 95% CI. Estimating the correlation and 95% CIs for the inflammatory agents and oxidative stress in the intervention group, compared with that in the control group (ALI), respectively (correlation: 0.635; 95% CI, 0.560–0.699, *P* value = 0.000, *Z* value= 12.648) and (correlation: 0.317; 95% CI, 0.189–0.434, *P* value = 0.00, *Z* value= 4.7). In conclusion, investigating the effects of different flavanols on oxidative stress in lung injury may provide a useful therapeutic strategy in ALI mouse models. However, the final conclusion on treatment efficacy should be sufficient for prospective controlled randomized trials.

Keywords Acute lung injury, Quercetin, Oxidative stress, Inflammatory responses, Lung injury model

Introduction

Acute lung injury (ALI) and acute respiratory distress syndrome (ARDS) are life-threatening lung diseases, and severe sepsis is one of the most important factors in causing the above diseases. However, it is not the only trigger that leads to ALI and ARDS. Factors such as severe bacterial pneumonia, trauma, exposition to injurious mechanical ventilation, and capillary endothelial cell damage are caused. ALI is associated with acute and severe inflammation that disrupts the endothelial and epithelial barriers of the lung. Alveolar-capillary membrane damage, pulmonary edema, neutrophil-induced inflammation, and perfusion-ventilation mismatch ultimately reduce lung compliance and cause profound hypoxemia and may lead to loss of lung function [1, 2]. However, drug treatments

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