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Research Article

Preventive Effects of Velvet Antler *(Cervus elaphus)* against Lipopolysaccharide-Induced Acute Lung Injury in Mice by Inhibiting MAPK/NF-κB Activation and Inducing AMPK/Nrf2 Pathways

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Velvet antler (Cervus elaphus) is a typical traditional animal medicine. It is considered to have various pharmacological effects including stimulation of the immune system, increase in the physical strength, and enhancement of sexual function. This paper aims to investigate the aqueous extract of velvet antler (AVA) in the mouse models of LPS-induced ALI. Inhibition of NO, TNF-α, IL-1β, IL-6, and IL-10 productions contributes to the attenuation of LPS-induced lung inflammation by AVA. A 5-day pretreatment of AVA prevented histological alterations and enhanced antioxidant enzyme activity in lung tissues. AVA significantly reduced the material (total number of cells and proteins) in the BALF. Western blot analysis revealed that the expression of iNOS and COX-2 and phosphorylation of IκB-α and MAPKs proteins are blocked in LPS-stimulated macrophages as well as LPS-induced lung injury in mice. Consistent with this concept, the phosphorylation of CaMKKβ, LKB1, AMPK, Nrf2, and HO-1 was activated after AVA treatment. The results from this study indicate AVA has anti-inflammatory effects in vivo and AVA is a potential model for the development of health food. In addition, its pathways may be at least partially associated with inhibiting MAPK/NF-κB activation and upregulating AMPK/Nrf2 pathways and the regulation of antioxidant enzyme activity.

1. Introduction

Acute lung injury (ALI) is the most common form of respiratory failure, which is shown by increasing capillary-alveolar permeability, changing in lung protein leaks, inflammatory cell accumulation, interstitial edema, disruption of the alveolar epithelium, excessive polymorphonuclear neutrophil (PMN) migration, and the release of proinflammatory cytokines and mediators in the lung [1]. This disease is associated with a great risk of morbidity and mortality in patients with shock, sepsis, ischemia-reperfusion, aspiration of gastric contents, major trauma, and other clinical disorders [2]. Macrophages can be activated by cytokines such as interferon-γ (IFN-γ) and lipopolysaccharide (LPS, bacterial endotoxin) and downregulation of proinflammatory cytokine release [tumor necrosis factor-alpha (TNF-α), interleukin-1β (IL-1β), and IL-6] and inflammatory factors (NO) that recruit additional immune cells to the tissue injury [3, 4]. Also, LPS promotes the production of reactive oxygen species (ROS) and reactive nitrogen species (RNS).