Received: 25 July 2019 | Revised: 24 December 2019 | Accepted: 27 December 2019

DOI: 10.1002/tox.22896

RESEARCH ARTICLE

ENVIRONMENTAL TOXICOLOGY WILEY

Resveratrol protects human bronchial epithelial cells against nickel-induced toxicity via suppressing p38 MAPK, NF-κB signaling, and NLRP3 inflammasome activation

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Funding information
National Natural Science Foundation of China, Grant/Award Number: 31770017; Startup Foundation for Doctors of Liaoning Province, Grant/Award Number: 20170520258; Scientific Research Fund of Liaoning Provincial Education Department

Abstract

Nickel is a common environmental pollutant that can impair the lung, but the underlying mechanisms have not yet been fully elucidated. Furthermore, natural products are generally used to inhibit cell damage induced by heavy metal. Resveratrol possesses wide biological activities, including anti-inflammation and antioxidative stress. This study was conducted to explore the toxicity of nickel on human bronchial epithelial (BEAS-2B) cells and evaluate the protective effect of resveratrol. The results showed that nickel could induce cell apoptosis, increase oxidative stress, and promote the expression of pro-inflammatory cytokines, including tumor necrosis factor-α, interleukin (IL)-1β, IL-6, IL-8, C-reaction protein. Western blot analysis showed that nickel activated p38 mitogen-activated protein kinase (MAPK), nuclear factor-kappa-B, and nucleotide-binding oligomerization domain-like receptor pyrin-domain-containing protein 3 pathways, while resveratrol could reverse these effects. Our results suggested that resveratrol could protect BEAS-2B cells from nickel-induced cytotoxicity. Therefore, resveratrol is a potential chemopreventive agent against nickel-induced lung disease.

KEYWORDS
inflammation, nickel, oxidative stress, resveratrol, ROS

1 | INTRODUCTION

Nickel is one of the metals widely found in the environment, food, chemicals and medicines.[1,2] The high consumption of nickel-containing products inevitably leads to the pollution of nickel.[3] The main sources of nickel exposure are the burning of petroleum and coal,[4] as well as nickel-related industries such as electronic circuits, glass coloring, and electroplating. The large amount of nickel may be deposited in the human lungs due to occupational or environmental exposure and causes tissue damage.[5] There has been evidence that nickel oxide nanoparticles (NiONP) can cause excessive production of intracellular reactive oxygen species (ROS), increase inflammatory cytokines in BEAS-2B cell and the inhalation of NiONP aggravates the inflammatory response in animals.[6,7] However, the possible mechanism of nickel-induced lung cell toxicity remains to be determined.

Some studies have revealed that the redox balance can be disturbed by excessive ROS which ultimately leads to severe lung tissue damage and disease progression.[8] Meanwhile, Zhang et al reported that ROS-mediated oxidative stress could be dramatically increased and activated specific redox-sensitive pathways, including p38 mitogen-activated protein kinase (MAPK), nuclear factor-kappa B (NF-κB) pathways which were vital to the cell apoptosis and inflammation.[9] Besides, nucleotide-binding oligomerization domain-like receptor pyrin-domain-containing protein 3 (NLRP3) inflammasome plays an important role in

Xiangyu Cao and Siqi Tian contributed equally to this study.

