

Abstract

The non-redox active transition metal Zn is a micronutrient that plays essential roles in protein structure, catalysis and regulation of function. Inhalational exposure to ZnO or Zn salts in occupational and environmental settings leads to adverse health effects, the severity of which appears dependent on the flux of Zn^{2+} presented to the airway and alveolar cells. The toxicity of exogenous Zn^{2+} exposure is characterized by cellular responses that include mitochondrial dysfunction, elevated production of reactive oxygen species and loss of signaling quiescence leading to cell death and increased expression of adaptive and inflammatory genes. Central to the molecular effects of Zn^{2+} is its interactions with cysteinyl thiols, which alters their functionality by modulating their reactivity and participation in redox reactions. Ongoing studies aimed at elucidating the molecular toxicology of Zn^{2+} in the lung are contributing valuable information about its role in redox biology and cellular homeostasis in normal and pathophysiology.