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The influence of atmospheric and cellular H2O2 on ROS concentrations and OH radical production in the lung

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Atmospheric pollution is a significant cause of oxidative stress in the human pulmonary system.(1–3) Fine particulate matter ($PM_{2.5}$) has been linked to adverse health effects due to the size and composition of particulates. Gas-phase chemical species, such as ozone, have also been considered as significant pollutants posing threat to human health.(3-4) However, other reactive gas-phase species, such as peroxides, have hardly been examined.

The epithelial lining fluid (ELF) is a large interface between the atmosphere and human body.(5) Transition metals enter the ELF through inhalation of $PM_{2.5}$ and play a key role in the potential occurrence of health effects. In the presence of transition metals, peroxides such as hydrogen peroxide (H_2O_2) are converted into the highly reactive OH radical through Fenton chemistry. Due to its high reactivity, the OH radical is most likely to cause oxidative stress and Fenton chemistry is probably an important OH source in the lung.(6) High levels in both, peroxide and transition metal concentrations in the ELF, could thus have adverse health outcomes.

We investigate the role of the most abundant atmospheric peroxide, H_2O_2, in the formation of reactive oxygen species (ROS: H_2O_2, OH, O_2^-, HO_2)(5) in the human body using a kinetic multilayer model. We find that, besides ambient concentrations, transport to and from lung cells and the circulatory system affects H_2O_2 levels in the ELF and, accordingly, exhaled breath condensate (EBC). The model predicts levels of H_2O_2 in EBC, lung cellular space, and blood, in agreement with the literature. The H_2O_2 concentration in the ELF, where measurements cannot be conducted easily, can be inferred from the model and used to estimate air pollution-induced ROS production in the human body. We present scenarios of atmospherically relevant conditions of H_2O_2 and $PM_{2.5}$ pollution in urban and rural areas and simulate the effect of co-inhalation of H_2O_2 and $PM_{2.5}$ on ROS production in the ELF. We discuss the hypothesis whether accumulation of H_2O_2, either by inhalation or in-body transport, may be a prerequisite for $PM_{2.5}$ toxicity

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