Fate of pathologically bound oxygen resulting from inhalation of labeled ozone in rats. Hatch, Gary E., Slade, Ralph and John McKee. Cardiopulmonary and Immunotoxicology Branch, Environmental Public Health Division, NHEERL, U.S. Environmental Protection Agency, Research Triangle Park, North Carolina, U.S.A. 27711.

ABSTRACT

Inhaled ozone (O_3) reacts chemically with respiratory tissues where it forms adducts with most biomolecules. We quantified the plasma concentrations and urinary excretion of ^{18}O in rats exposed to $^{18}O_3$ in order to gain insight into O_3 injury and repair. Male Fischer 344 rats were exposed to $^{18}O_3$ (2 ppm, 6 hr or 5 ppm, 2 hr) and urine was collected twice daily for 4 days. Increased ^{18}O was detected in dried blood plasma at 7 hr post exposure and in dried urine for at least 4 days post exposure with higher excretion rates at night. Total ^{18}O excreted was ~ 53% of the estimated amount of $^{18}O_3$ retained by the rat during $^{18}O_3$ exposure. Urinary ^{18}O appeared to be of respiratory origin (not ingested), it was enriched in the >500 MW fraction, and it was stable to 250°C. Pre-exposure to unlabeled O_3 a week earlier did not alter the excretion of ^{18}O into urine following $^{18}O_3$ exposure. We conclude that reaction products originating from $^{18}O_3$ inhalation enter the circulation and are excreted into urine with only moderate recycling.

DISCLAIMER

The research described in this article has been reviewed by the National Health and Environmental Effects Research Laboratory, United States Environmental Protection

Agency and approved for publication. Approval does not signify that the contents necessarily reflect the views and policies of the agency, nor does mention of trade names or commercial products constitute endorsement or recommendation for use.

KEYWORDS

Ozone

Oxidative stress

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Adducts

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INTRODUCTION

Ozone (O_3) pollution of ambient air has been shown to have worldwide health effects including epidemiological associations with cardiopulmonary endpoints;[1],[2-4]. Animal studies have shown that O_3 can affect extrapulmonary sites such as enhancement of atherosclerotic plaques and vascular injury in susceptible animals [5,6]. Due to its low water solubility and high chemical reactivity, O_3 is able to penetrate deeply into the respiratory tract where it forms stable adducts as well as reactive intermediates such as peroxides, aldehydes and carbonyls [7,8]. Inhaled O_3 in animals causes oxidation of