

EXECUTIVE SUMMARY

THE INFLUENCE OF EXERCISE ON LUNG INJURY INDUCED BY
OZONE AND NITROGEN DIOXIDE

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The purpose of this research was to examine factors modifying lung lesions induced in laboratory rats by ozone inhalation. An earlier study (ARB Contract A0-129-32) demonstrated enhancement of lung damage by light exercise; in exposures with intermittent rest and exercise, ozone concentration and the proportion of time spent in exercise were important factors increasing abundance and severity of lung lesions. For the present study a rat exercise protocol was developed to utilize continuous exercise at higher workloads. In terms of the increase in metabolic gas exchange over resting rates, the moderate exercise levels in this study were analogous to human walking or light manual labor. Lung lesions were quantified as percent of parenchymal cross section area involved for two morphological types of lesions: Type I: free cells in alveolar spaces, and Type II: alveolar duct walls and alveolar septa thickened due to infiltrating cells. Three experiments were performed: 1) Effect of duration of exposure to 0.35 ppm O₃ during exercise, 2) Effect of increasing exercise workload at constant effective dose of 0.35 ppm O₃, and 3) Effect of 0.35 ppm O₃ and 0.6 ppm NO₂ inhaled alone and in combination during exercise. In the test of exposure duration, rats were exposed to 0.35 ppm O₃ while running 15 m/min at 20% grade for 0.5, 1.0, 2.0, or 3.0 hours. Type I lesion areas did not differ from control (1.8% of parenchymal section area) for 0.5 and 1.0 h exposures but increased by nearly a factor of 2 (3.4% and 3.6%) in 2.0 and 3.0 h exposures respectively. Type II lesions were first apparent in the 1.0 h exposure (0.18%) and increased to 2.6% and 3.2% in 1 and 3 h exposures.

In the second experiment, rats were again exposed to 0.35 ppm O₃. Minute ventilation could not be measured to accurately calculate effective dose of O₃, however oxygen consumption was measured and used as an index proportional to ventilation. Exposure duration was adjusted to hold the quantity (ppm O₃)·(duration)·(average \dot{V}_{O_2}) constant. Exercise levels and corresponding exposure durations were continuous rest (3.4 h), run 8 m/min at 0% grade (2.75 h), 15 m/min at 20% grade (2.33 h), and 30 m/min

at 20% grade with 2 rest periods of 7 min (1.75 h). Despite near equivalence of effective dose of O₃, lesion areas increased with increasing exercise intensity. Type I lesion areas for rest and 8 m/min, 0% grade groups were similar to control, but increased by factors of 1.5 and 3 (to 3.2% and 6.4% of parenchymal area) for exercise at 15 m/min, 20% grade and 30 m/min, 20% grade respectively. Type II lesions were present at low incidence (0.15% of parenchymal area) in the rest exposure and progressively increased by factors of 3 (to 0.5%), 16 (to 2.4%), and 33 (to 5.0%) for the 3 successive exercise workloads.

In the O₃ and NO₂ interaction experiment, rats ran at 15 m/min 20% grade for a 3 hour exposure. Exposure groups were 0.35 ppm O₃, 0.6 ppm NO₂, and combination 0.35 ppm O₃ and 0.6 ppm NO₂. Exposure to NO₂ alone resulted in no difference from clean air control, however NO₂ in combination with O₃ resulted in enhancement of lesion areas induced by O₃ alone. The experiment was repeated, and the results were similar. Combined results of the experiments showed Type I lesion areas induced by O₃ alone were increased above control by a factor of 1.6 (to 2.9% of parenchymal area), but in combined O₃ and NO₂, lesion areas increased by a factor of 2.7 over control (to 4.8% of parenchymal area). Type II lesion areas (not found in control clean air exposure) induced by exposure to O₃ alone (2.2% of parenchymal area) were increased by a factor of 2.3 (to 5.1%) when NO₂ was present as a co-pollutant.

Results of these experiments demonstrated the critical importance of common modifying factors such as exercise and co-pollutants to damage to the respiratory system. Although it is not likely that rats and humans develop the same quantitative extent of tissue damage from oxidant exposures, the qualitative effects and relative importance of the exposure variables tested (exercise and co-presence of NO₂ with O₃) are expected to be similar. Furthermore, sensitivity of both humans and animals is highly variable, and the range of human sensitivity may overlap that of the rat model.

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