Effect of Exposure to 43 ppm Nitric Oxide and 3.6 ppm Nitrogen Dioxide on Rabbit Lung

A Light and Electron Microscopic Study*

C. Hugod

Department of Clinical Chemistry, Rigshospitalet, Blegdamsvej 9, DK-2100 Copenhagen, Denmark

Summary. Continuous exposure of rabbits to 43 ppm nitric oxide and 3.6 ppm nitrogen dioxide for 6 days did not cause morphological changes in the lungs when compared to specimens from nonexposed rabbits. This is not in accordance with previously reported findings.

Key words: Nitric oxide (NO) — Nitrogen dioxide (NO$_2$) — Rabbit lung — Histo-toxicity — Artefacts

Although nitric oxide (NO) occurs in concentrations up to 1000 ppm in tobacco smoke, it has mainly attracted toxicological interest because of its role as 'primary pollutant' in the formation of photochemical smog. In a recent report, results from a 14-days-lasting exposure study of rabbits to 5 ppm NO were reported, demonstrating electron-lucid vacuoles inside the arteriolar endothelial cells and/or the intercellular junctions and thickening of the alveolo-capillary membrane possibly due to accumulation of edema fluid in the interstitial space [6].

For further investigation of the toxic effect of NO on lung tissue the present experiment was performed. Attempting to work with NO as the only air pollutant, the inevitable contamination with the far more toxic oxidation product nitrogen dioxide (NO$_2$) must be accounted for. Two methods to avoid this contamination have been described in the literature. Von Nieding [8] trapped the NO$_2$ in an alkaline solution of a mixture of sulfonic acid and salicylic acid, while Oda [9] used soda lime. Both methods were concurrently applied in the present experiment.

Materials and Methods

Twelve male rabbits (Danish country breed) were used. Weight: 2.0–2.5 kg.

The experimental group consisted of 6 rabbits being exposed as described below. The control animals were breathing atmospheric air, but were kept under otherwise identical conditions. The animals were fed standard rabbit pellets and tap water ad libitum.

* Supported by a grant from Commission of the European Communities, Environmental Research Programme, Contract No.: 031–74–1 ENV DK

0304–0131/79/0042/0159/$ 1.80
Fig. 1a Pulmonary arteriole demonstrating vacuolisation of endothelium. Phase contrast x 245.
b Pulmonary vessel demonstrating slight vacuolization of endothelium and focal areas with widening of the subendothelial space. Phase contrast x 147