613 MODULATION OF OZONE ABSORPTION BY INTERFACIAL PHOSPHOLIPIDS.

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Reactions of ozone (O_3) and nitrogen dioxide (NO_2) with constituents of the lung surface lining layer mediate the net flux of inhaled oxidant into the aqueous phase (reactive absorption). Recent studies have shown that monolayers of dipalmitoyl phosphatidylcholine (DPPC), the major component of alveolar surfactant, modulate NO_2 uptake. When DPPC, deposited on an aqueous surface, was subjected to increased molecular packing (surface compression), NO_2 reactive absorption was significantly reduced. To investigate potential phospholipid (PL) induced effects on O_3 uptake, the rates of O_3 gas phase disappearance (25° C) were evaluated using a specialized exposure-compression apparatus. Determinations of O_3 mass balance across a defined surface area were conducted during graded PL surface compressions. DPPC, dilinoleoyl-PC (DLPC) or palmitoyloleoyl PG (POPG) monolayers were compressed from 121 → 49 Å^2/molecule, γ = 72 → 25 dyn/cm respectively. Apparatus constraints prevented further compression. **Results:** 1) At the initial area/molecule level, PL did not alter O_3 (0.6 ± 0.1 ppm) absorption rates. 2) Upon DPPC compression, uptake declined proportional to surface compression (3.5 → 1.2 μg O_3•min^{-1}•cm^{-2}). 3) DPPC-induced reductions occurred regardless of aqueous substrate concentration, composition, or [O_3] (0.3 to 1.0 ppm). 4) Compression of POPG or DLPC monolayers did not significantly decrease uptake. 5) DLPC, extracted from O_3-exposed liposomes, when compressed displayed similar surface tensions and O_3 absorption as previously unexposed. **Conclusions:** Compression of DPPC monolayers to surface tensions which are still greater than those occurring *in vivo* significantly limits O_3 flux into the aqueous phase. Under the exposure conditions employed in these studies, it is unlikely that O_3 reactions with interfacial phospholipids contribute significantly to absorption, nor do such reactions appear to alter PL surface active properties. (HL54696, ES05749, and HL24075)

614 ADHESION MOLECULES ON BLOOD NEUTROPHILS AND ALVEOLAR MACROPHAGES FROM RATS: MODULATION BY EXPOSURE TO OZONE.

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It is known that exposure of rats to the pulmonary irritant ozone results in an infiltration of polymorphonuclear leukocytes (PMNs) into the lungs. The purpose of this study was to investigate whether the ozone-induced inflammatory process is preceded by a change in the expression of integrins and selectins in peripheral blood PMNs and alveolar macrophages. Female Sprague Dawley rats were exposed to air or ozone (1 ppm, 2 or 4 hrs). Bronchoalveolar lavage (BAL) was carried out and blood was collected via intracardiac puncture at 0 or 18 hrs after the exposure. The percent of neutrophils in the BAL fluid was increased only in animals exposed to ozone for 4 hrs and not for 2 hrs, while the expression of CD18 on alveolar macrophages was lowered at all exposure times. The expression of CD62L on blood PMN was not affected by exposure to ozone, while the expression of CD11b was lowered after 2 hrs, and not after 4 hrs of exposure to ozone. This trend was also observed in experiments in which plasma of ozone-exposed animals was incubated with whole blood obtained from non-exposed animals. In these experiments, the expression of CD11b on PMNs of non-exposed animals was lower after incubation with plasma from 2 hours-ozone-exposed animals, but not after incubation with plasma from 4 hours-ozone-exposed animals. (This work was supported by a grant from the Committee on Prevention and Research in Occupational Health at the Ministry of Labour and Social Welfare.)

615 ACUTE INFLAMMATORY REACTION IN RATS AFTER INTRATRACHEAL INSTILLATION OF MATERIAL COLLECTED FROM A NYLON FLOCKING PLANT.

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The etiologic agent responsible for several cases of interstitial lung disease among workers at a nylon flock plant was unknown. Airborne dust collected at the plant was examined for its inflammatory potential in rat lungs. The endpoints measured were: (1) breathing rates, (2) differential cell counts of bronchoalveolar lavage cells, (3) alveolar macrophage (AM) chemiluminescence, (4) albumin in the acellular bronchoalveolar lavage, and (5) pulmonary histopathology. In the first study, rats received a single dose of the airborne dust sample (10 mg/kg body weight) by intratracheal (IT) instillation. At 1 day post-IT, all inflammatory endpoints were significantly increased ($p<0.05$) versus controls but by 29 days post-IT they did not differ significantly ($p>0.05$) from controls. Histopathology demonstrated mild to moderate, multifocal, suppurative pneumonia usually centered around bronchioles at 1 day post-IT. At 29 days post-IT, pulmonary inflammation was minimal to mild and characterized by alveolar histocytosis usually restricted to the immediate area of retained birefringent fibers. In subsequent experiments, airborne dust was extracted with water and the dust (washed airborne dust) and water extract (soluble fraction) were separated by centrifugation. Nylon dust was prepared in the laboratory by milling uncut nylon strands that had not been treated with finish or dyes which are commonly used in nylon flock plants. Rats were administered a single dose of a dust sample (10 mg/kg body weight) or the soluble fraction (1.3 ml/kg body weight) by IT administration and the same endpoints were measured at 1 day post-IT. The dust samples caused significant increases in all of the inflammatory end points; however the soluble fraction was by far the least active. Histological analysis of the lungs 1 day post-IT confirmed lung inflammation was occurring only after dust instillation and tended to center around bronchioles. The results suggest: (1) nylon flocking generates particles of respirable size which can interact with AM and can be detected in the lung 29 days after exposure, (2) the dusts examined cause an inflammatory response, (3) water extractable agent(s) from airborne dust contribute minimally to the inflammatory response, and (4) the acute inflammatory response to these dusts is substantial when compared to other pathologic occupational dusts previously examined.

616 INTRATRACHEAL INSTILLATION OF WELDING FUMES ALTERS THE PULMONARY CLEARANCE OF *LISTERIA MONOCYTOGENES* IN THE RAT.

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Epidemiological studies have shown an increased incidence of respiratory illness in welders. Stainless steel (SS) welding fumes have been shown to have a greater effect on alveolar macrophage (AM) function than mild steel (MS) fumes. The objective of this study was to evaluate the effects of different welding fumes on the clearance of a bacterial pathogen from the lungs. Fumes were collected during flux-cored manual metal arc (MMA) and gas metal arc (GMA) welding using either SS or MS consumable electrodes. The fume composition was: **1) GMA-SS**: 52.3% Fe, 22.2% Cr, 18.3% Mn, 4.9% Ni, 2.3% Si; **2) GMA-MS**: 89.2% Fe, 8.2% Mn, 2.6% Si; **3) MMA-SS**: 22.3% K, 19.4% Fe, 13.1% Cr, 12.6% Si, 8.2% Ca, 8.0% Mn, 17.4% other. CD/VAF rats were dosed intratracheally with saline (control) or the welding samples at a dose of 1.0 mg/100 g b wt. At 1 and 35 days post-instillation, 5000 *Listeria monocytogenes* were intratracheally instilled into the treated animals. Five days after intratracheal exposure to *L. monocytogenes*, the lungs and spleen were removed, homogenized, and cultured quantitatively on Brain Heart Infusion agar at 37° C. Colony forming units (CFUs) were counted after an overnight incubation. After a 1 day fume treatment, all three welding samples caused a significant decrease ($p<0.05$) in the 5-day clearance of *L. monocytogenes* from the lungs as compared to control. At 35 days post-instillation, the MMA-SS and GMA-SS samples significantly increased ($p<0.05$) *L. monocytogenes* clearance from the lungs as compared to the GMA-MS and control groups. No significant differences were seen among the treatment groups in the number of bacteria cultured from the spleen. We have demonstrated that subchronic exposure to SS fumes may have a greater effect on the pulmonary clearance of *L. monocytogenes* as compared to MS fumes. This is most likely due to differences in fume composition and the greater effect of SS fumes on AM function.

617 APOPTOSIS INDUCTION AFTER SILICA INHALATION IN RATS.

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Occupational exposure to crystalline silica (quartz) has been associated with lung damage and fibrosis. In vitro studies have shown that silica induces apoptosis in human alveolar macrophages and in vivo after intratracheal

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Preface

This issue of *The Toxicologist* is devoted to the abstracts of the presentations for the symposium, platform, poster / discussion, workshop, roundtable, and poster sessions of the 38th Annual Meeting of the Society of Toxicology, held at the Ernest N. Morial Convention Center, New Orleans, Louisiana, March 14-18, 1999.

An alphabetical Author Index, cross referencing the corresponding abstract number(s), begins on page 419.

The issue also contains a Keyword Index (by subject or chemical) of all the presentations, beginning on page 444.

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