

**[Poster Board #B23] Oxidative Stress in Acute Silicosis: Correlation with Toxicity and Oxidative Stress,
[Publication Page: A656]**

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In vivo electron spin resonance (ESR), O₂ uptake, and whole body imaging were used to investigate the toxicity responses and oxidative stress associated with the development of acute silicosis. Mice were exposed by aspiration of 70 µg of silica in 25 µL of 0.9% NaCl. Control and experimental animals at 1, 3, and 7 days post exposure had broncho-alveolar lavage (BAL) and blood drawn for bioassays. The ESR measurements and whole body imaging were performed with a separate group of mice. Bioassays included measurements of albumin, protein, lactate dehydrogenase (LDH), and N-acetylglucosaminidase (NAG) in BAL fluids. Silicosis-induced secondary signals for IL-1, TNF-α, total antioxidant capacity, catalase, SOD and glutathione also were measured in the serum. ESR and imaging measurements were performed after i.p. of TEMPOL or 3-CP nitroxide at a final concentration of 344 mg/kg body weight. LiNC particles were implanted into the mouse thigh to measure pO₂ levels after exposure. Albumin, protein and NAG levels increased significantly in BAL fluid post 3 day exposure implying damage caused by silica in the lung. Impairment in the ability of silica exposed animals to clear radicals during acute silicosis was evident at days 1 and 3 post exposure. ESR imaging provided information on the location and distribution of the 3-CP label within the lungs and heart and its impaired clearance. Mice with the implanted LiNC particle showed decreased pO₂ levels in correlation with these bioassays post 3 days. Bioassays in concert with ESR and imaging presented in this study provide congruent data on the early acute phase of pulmonary injury and a decline in oxidative stress in response to acute silicosis.

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Session Info: [] Thematic Poster Session, [C131] GLOBAL OCCUPATIONAL AND ENVIRONMENTAL DISEASES**

Session Time: 8:15 AM - 4:15 PM

Presentation Date: Tuesday, May 23, 2006

Presentation Time: 8:15 AM

Room: Area B (Sails Pavilion, Upper Level), San Diego Convention Center

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