those of EU. We consider that presented data must be an alarm signal for organizers and workplaces managers in other to remedial the situation with negative impact upon health of employees, the presentation to the medical prophylactic examinations represents a way of finding incipient discover of illness for some entities sometimes irreversible.

343 Silicotics in Germany - mortality risk and exposure level

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Background: In order to prevent lung cancer among silica exposed workers a safe level needs to be established.
Method: In a prospective cohort study 630 silicotics (compensated between 1988 and 2000) from three different industries (stone & quarry, ceramics and tunnelling) have been enrolled.
Result: Within the follow-up over 212 of the workers died. The overall as well as the lung cancer mortality rate is about twice as high as in the general population. All workers have been exposed for at least for one year above the current MAK-value of 0.15 mg/m³. The cumulative exposure has been 2 mg/m³ year or greater. All workers have been exposed on average about 33 years.
Conclusion: This study shows the high mortality risk for compensated silicotics. As result of this study, the exposure has to be at a level of 0.15 mg/m³ at any time or lower. The latency period is too short to give additional information about a "safe" level, e.g. whether a value of 0.15 mg/m³ is sufficient in order to prevent silica induced lung cancer.

345 Mechanisms of crystalline silica and coal-induced emphysema development

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Background: The incidence of chronic obstructive pulmonary disease (COPD) has increased dramatically in the past few decades and now ranks as a major cause of morbidity and mortality on a worldwide basis. It is estimated that approximately 14 million people in the United States have COPD. The World Health Organization predicts that by 2020 COPD will become the 3rd most common cause of death. Cigarette smoking, environmental pollution, and occupational exposure are the most important risk factors associated with the development of COPD. Although COPD encompasses chronic obstructive bronchitis and emphysema the molecular mechanisms involved in their pathogenesis and expression of symptoms are dramatically different between these two diseases. Therefore, we will be focusing only on the potential mechanisms of silica or coal-induced emphysema development. In epidemiologic and pathologic studies occupational exposure to crystalline silica and coal are two important risk factors identified to be associated with the development of emphysema. Development of focal emphysema in coal miners who have never smoked is induced by the secretion of proteolytic enzymes from coal-activated macrophages and inactivation of α_1 antitrypsin. Similarly emphysema in silica-exposed workers is thought to be induced by the enhanced generation of reactive oxygen species secreted by activated macrophages and the resultant inactivation of α_1 antitrypsin. This hypothesis is supported by epidemiological studies documenting exposure-response for air flow obstruction in silica-exposed workers even in the absence of radiological signs of silicosis. In addition, *in vitro* and *in vivo* experimental studies corroborate the oxidative inactivation of antiproteinases and the subsequent breakdown of connective tissue in a dose-response fashion due to exposure to silica or coal. These experimental studies and other clinical observations suggest that silica-and coal dust-induced emphysema occur through similar mechanisms as that for smoking-induced emphysema.
Methods: In this report, we will review the epidemiologic and pathologic evidence and discuss the molecular mechanisms relevant to the development of emphysema in silica and coal-dust exposed workers within the context of recent findings.
Results and Conclusions: The evidence surveyed suggests that chronic exposure to silica or coal that may cause the development of chronic bronchitis, emphysema and/or small airways disease that can lead to airflow obstruction even in the absence of radiological disease.

346 Positron Emission Tomographic (PET) imaging of silicosis in a rabbit model using an ^{18}F-Fluorinated proline amino acid analog tracer

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The sensitivity of positron emission tomography using a proline amino acid analog was tested for detection of pulmonary response in early silicosis in an animal model subject to an acute instillation exposure to quartz dust. *Cis*-4-^{18}F fluoro-L-proline (FP) was synthesized as a positron-emitting ^{18}F-labeled analog of the amino acid proline. Proline and glycine are used extensively in procollagen synthesis by pulmonary fibroblasts in active silicosis. Proline is taken up by the fibroblasts to provide both the proline and hydroxyproline residues of procollagen, which is then secreted into the extracellular space for maturation into collagen scar tissue in fibrosis. ^{18}F is covalently bound in the FP proline analog. ^{18}F has an 110 minute half-life and decays by positron emission, finally resulting in emission of two 0.511 MeV gamma rays which provide a means to map the location of the tracer. Most clinical PET imaging uses an analog of sugar, fluorodeoxyglucose (FDG), to map areas of generally heightened metabolism, e.g., tumors. Instead, fluoroproline was tested here for above-background uptake associated with heightened metabolism specific to silicosis. Rabbits were instilled with respirable quartz dust in saline, or with saline alone for controls, and subsequently at 1, 2, 4, or 5 months animals were injected with 1mCi of FP and imaged in a clinical PET system. Animals were killed two days later and their lung tissue

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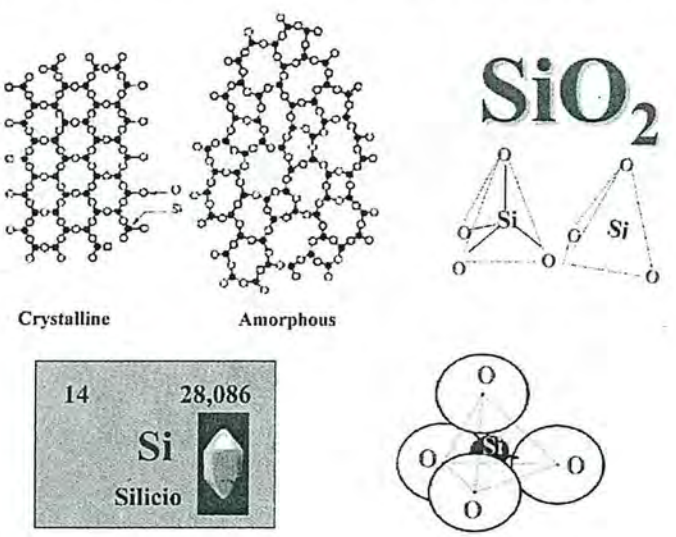

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