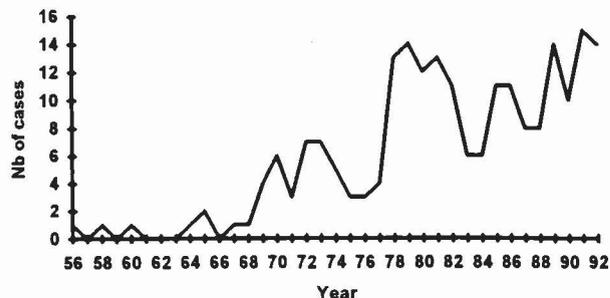


MALIGNANT PLEURAL MESOTHELIOMA IN NANTES-SAINT NAZAIRE AREA (FRANCE) 1956-1992. E Chailleux, D Pioche, S Chopra, G Dabouis, P Germaud, AY de Lajarte, M de Lajarte. Services de Pneumologie de Nantes et Saint Nazaire, France.

The existence of large shipyards accounts for the particular frequency of pleural mesothelioma in the department of Loire-Atlantique, on the Loire River estuary. The purpose of the present study was to note all of the cases diagnosed in the department between 1985 and 1992 in order to update earlier studies (Chest 1988;93:159-62) and to evaluate the trend in incidence. The list of cases was taken from the files of the Pathology Laboratories. Data concerning patients were gathered retrospectively from medical files of hospital departments and specialists. From 1955 to 1984, 125 cases (119 men, 6 women) were diagnosed and 91 from 1985 to 1992 (78 men, 13 women). The incremental trend in yearly incidence is highly significant (Spearman's rank correlation test). Mean age of subjects was rising during the studied period (59.1 ± 9.3 in 1956-1972, 61.5 ± 11.3 in 1973-1982 and 67.5 ± 10.0 in 1983-92). Occupational exposure to asbestos was certain or probable in 88% of cases, with a median length of exposure of 24 years (range one month to 48 years), and a mean interval between the beginning of exposure and the diagnosis of 43.5 ± 11.3 years (range 10 to 70). The industrial sector most often involved was shipbuilding (117 cases). The mean incidence in Loire-Atlantique during the period 1985-92 was 10.8 per million (19.1 for men, 3.0 for women). The male incidence were particularly high in the Saint Nazaire area (39.6 per million).



In conclusion our study confirms that the incidence of mesothelioma is still rising in Loire-Atlantique and is one of the highest reported in France.

IS LUNG CANCER RELATED TO ASBESTOS EXPOSURE IN THE ABSENCE OF PULMONARY FIBROSIS? A CASE-REFERENT STUDY

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Whether asbestos can cause lung cancer without inducing radiologically apparent fibrosis is an important scientific, practical and medico-legal question, but the epidemiological evidence is inconclusive. To investigate this further a case-referent study was carried out at the London Chest Hospital between September 1991 and March 1993. Detailed occupational and smoking histories were obtained from 270 patients with confirmed primary lung cancer, 276 referents with respiratory disease and 404 with cardiac disease. These histories were reviewed blind and in detail to assess the timing, duration and probability of exposure to asbestos and other selected agents. The presence and extent of fibrosis was assessed from chest radiographs, suitably obscured to avoid bias from evidence of the primary diagnosis. All films were scored by three expert readers blind and independently for small opacities and other changes using the ILO classification for pneumoconioses. The frequency, duration and intensities of asbestos exposure were based on detailed assessment of work histories. Ninety four (35%) of the lung cancer patients had worked in occupations categorized as having definite or probable asbestos exposure at least 15 years before diagnosis against 174 (26%) of controls (crude odds ratio 1.49; 95% CI 1.09-2.02). When the analysis was confined to subjects with median ILO scores of less than 1/0 the crude odds ratio (95% CI) was reduced to 1.21 (0.84-1.73), but when adjusted for age, sex and smoking habit the odds ratio was 1.64 (1.06-2.53). These and more detailed analyses suggest that workers from occupations with a high probability of exposure to asbestos are at increased risk of lung cancer even in the absence of radiological evidence of pulmonary fibrosis.

This project was supported by a grant from the Health & Safety Executive (UK).

LUNG FUNCTION ABNORMALITIES AMONG LUMBER MILL WORKERS WHO MAINTAIN TUNGSTEN CARBIDE TIPPED SAWS. Kennedy SM, Chan-Yeung M, Teschke K., UBC Dept of Medicine and Health Care and Epidemiology, Vancouver, Canada.

Hard metal lung disease is associated with cobalt in tungsten carbide tool manufacturing, but has only recently been reported among workers maintaining these tools. We report here results of a study of lumber mill saw filers who maintain tungsten carbide tipped saw blades. We studied cobalt exposure levels and determinants of exposure at 8 lumber mills, and tested lung function and symptoms in 118 saw filers at these mills (90% participation). Methods included a standardized questionnaire, spirometry, personal air sampling (278 samples over 4 months), and detailed examination of tasks performed every 10 minutes. Results were compared to data from bus mechanics tested using similar methods. Average cobalt exposure levels were low (mean: $5 \mu\text{g}/\text{m}^3$) but increased cobalt exposure was associated with tasks involving tungsten carbide grinding. Saw filers reported a 2 fold increase in phlegm and wheezing ($p < .01$) and a 3 fold increase in work-related cough, phlegm, and wheezing ($p < .001$), but no increase in breathlessness. Saw filers doing tungsten carbide wet grinding (ie. using coolant more than 50% of the time) had significant reductions in FEV_1 and FVC ($p < 0.05$) and were more likely to have FEV_1 and FVC values less than 80% of predicted (odds ratios: 6.6 for FEV_1 , $p < .05$; and 5.2 for FVC, $p < .10$). Linear regression analysis indicated that both cobalt exposure level and duration of work involving tungsten carbide grinding were associated with significant reductions in FEV_1 and FVC. Further analysis suggested that the effect of cobalt exposure level on lung function was most pronounced in association with coolant lubricated tungsten carbide grinding. No lung function abnormalities were seen among workers grinding other metal alloys using these same coolants. As average cobalt exposure levels were well below the currently accepted permissible concentration, these findings suggest that the current exposure limit for cobalt may be too high; and further, that it is possible to reduce exposures to levels well below the current limit.

WHOLE LUNG LAVAGE FOR THE TREATMENT OF SILICOSIS. JL Wilt, DE Banks, NL Lapp, DN Weissman, HV Dedhia, JE Parker, JKH Ma, E Stullken, RP Benzo, H Huang, V Vallyathan, V Castranova, J Shumaker., Section of Pulmonary and Critical Care, Department of Medicine and Anesthesiology, West Virginia University, and NIOSH, Morgantown, WV.

Silicosis is an irreversible, chronic, progressive respiratory disease. No therapy has been proven to effectively modify the progression of silicosis. Whole lung lavage (WLL) is used in treating Pulmonary Alveolar Proteinosis but reported only once in the Western literature as treatment for dust-induced lung disease. Recently physicians in China have utilized this therapy for pneumoconiosis in coal miners. The rationale for WLL in dust-induced lung disease is removal of activated cells, cytokines and dust-containing cells, all of which are likely contributing to the fibrosis. We performed WLL on a 53 year old motorman and roof bolter with significant silica exposure, ILO category 2 with coalescence chest radiograph, and normal pulmonary function. We also performed a segmental bronchoalveolar lavage (BAL) prior to and 10 days after the WLL on each side. WLL on the right lung consisted of 8 liters of saline, at which time he suffered a hydropneumothorax which led to premature termination of the procedure. A small catheter was inserted, the pneumothorax evacuated, and he was discharged the following day. Six weeks later, he underwent WLL on the left with 13 liters of saline without incident. Total cells recovered during WLL were 5.22×10^8 on the right and 3.91×10^8 on the left. Total macrophages recovered were 4.71×10^8 on the right (90% of total cells) and 3.70×10^8 on the left (94.6% of total cells). WLL removed 1.82 gm of mineral dust (non-coal) on the right and 1.64 gm on the left. Cytokine analysis paralleled the dust and radiographic findings of more disease on the right. Cultured alveolar macrophages from pre-WLL lavage released higher basal $\text{TNF}\alpha$ values on the right (213 pg/ml) versus left (28 pg/ml). With zymosan stimulation the levels increased to 331 pg/ml (right) and 213 pg/ml (left). Similar values were obtained for basal $\text{IL-1}\beta$, 322 pg/ml (right) and 18 pg/ml (left). Zymosan stimulated cells yielded 396 pg/ml from the right versus 115 pg/ml from the left lung. WLL appears to be a feasible means of removing dust, cytokines, and dust-laden cells that may reflect differential disease activity and allow modification of the progressive fibrosis that is associated with silica exposure.

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