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EMPHYSEMA IN COAL WORKERS' PNEUMOCONIOSIS: CONTRIBUTION BY COAL AND CIGARETTE SMOKING. FHY Green, V Vallyathan, P Schleiff, and M Attfield. Div. of Resp. Dis. Studies, NIOSH, Morgantown, WV 26505 USA.

These studies were undertaken to determine and differentiate the relationship of age, coal mining tenure, cigarette smoking, and coal workers' pneumoconiosis (CWP) to tha type and severity of emphysema found in a group of autopsy cases from southern West Virginia. The study group consisted of 266 coal miners (41 non-smokers) and 75 non-miner controls (14 non-smokers). Whole lung sactions prepared systematically were reviewed by two pathologists and emphyaema and CWP were classified and graded for severity using a standardized grading schema. The results of these studies showed no correlation between age and emphysema index in non-miner, non-smoker controls (r=0.15), but the non-miner controls who smoked showed a strong correlation to years smoked (r=0.63) and increasing age. In the non-smoking coal miners, emphysema was significantly higher than in controls ($p < 0.01$). Emphysema index in the coal miners who smoked also showed a significant increase compared to non-miner smokers ($p < 0.01$). Coal mining tenure also showed a significant relationship with emphysema index for both smokers and non-smokers ($p < 0.01$). An additive effect of cigarette smoking and coal mining was evident. The most common type of emphysema in non-miner controls who smoked was centriacinar, and in coal miners with and without cigarette smoking exposure, it was focal emphysema associated with the macule, indicating that both cigarette smoking and coal mine dust exposure preferentially affect the centriacinar portion of the lobule. Panacinar emphysema was found only in the smokers. There was a slightly positive relationship between the ratained coal dust in the lung and the emphysema index ($p = 0.05$). The results of these studies indicate that coal dust and cigarette smoking play important roles in the development and severity of emphysema in coal miners.

GENE EXPRESSION OF TYPE I AND III COLLAGEN AND REGULATION DURING EXPERIMENTAL SILICOSIS AND ASBESTOSIS IN RATS

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Silicosis and asbestosis are two kinds of occupational fibrotic diseases .The pathogenic mechanisms are still incompletely understanding, We assume that increase of gene expression of collagen Type I and III may be contributed to their molecular mechanism.In this study, (1)Dot blot and in situ hybridization methods were employed to evaluate the changes of Type I and III collagen gene expression during experimental silicosis and asbestosis in rats. (2)The regulatory effect of a fibrogenic factor on collagen gene expression in 2BS cells were studied in vitro. cDNA probes of collagens were prepared from Hf677, Hf322 and Sp6 α 1(III) recombined plasmids which would be complimentory to the 3'- terminal nuclectides of pro α 1(I), pro α 2(I) pro α 1(III) collagen mRNA respectively. The fibrogenic factor was derived from alveolar macrophages of silicotic rats with a molecular weight of 66KD which was different from other factor reported in literatures. The results showed that there were increases of both Type I and III collagen gene expression in experimental silicosis and asbestosis, but the peaks of increase of these two types of collagen gene expressions appeared at different periods. In vitro test demonstrated that chrisotile and crocidolite could stimulate the collagen gene expression. The additive stimulating effect of asbestoes and the fibrogenic factor was higher than any of the single factors. Actinomycin D may inhibit the stimulation. The above results showed that molecular hybridization methods are sensitive and applicable for studying of mechanism of pneumoconiosis.

Key words: Silicosis Asbestosis Collagen gene expression

CROSS-SHIFT CHANGE IN FEV1 AS A PREDICTOR OF SUBSEQUENT LONGITUDINAL DECLINE IN FEV1 AMONG GRAIN ELEVATOR WORKERS. SM Kennedy, H Ward, M Chan-Yeung. UBC Dept of Medicine, Vancouver, Canada

Some grain elevator workers experience an acute drop in FEV1 over one work shift. It has been suggested that this may be an indicator of an increased risk for subsequent more chronic lung function impairment. Cross-shift change in apirometry was measured on all available workere from 5 Port of Vancouver grain elevators in 1975. Subsequent cross-sectional surveys were conducted at these elevators approximately every 3 yeara. In 1990, retired membera of the population were invited to participate in a further survey (participation rate: 65%). Of the 82 retired grain elevator workers studied in 1990, 61 had performed the cross-shift apirometry test in 1975 and had at least one additional measurement of lung function between 1978 and 1990. We calculated longitudinsl dacline in FEV1 for theee workers as tha least squares slope of all available FEV1 data from surveys between 1978 and 1990. The 1975 data were only used for the daterminstion of acute shift change. 12 of the 61 retirees had a 5% or greater acute work-shift decline in FEV1 in 1975 (responders). The acute responders did not differ from the remainder with respect to age, height, weight, smoking status, or amount amoked; they were more likely to be atopic (16% vs 4%). 1975 lung function valuee (as % of predicted) for the respondera vs non-responders were 84% vs 88% (p=.5) for FEV1, 93% vs 92% (p=.9) for FVC, and 70% va 87% (p=.09) for MMF. 1990 lung function values for these groups were 67% vs 83% (p<.05) for FEV1; 84% vs 92% (p=.2) for FVC; and 39% vs 64% for MMF (p<.05). Multiple regression analysis with subsequent longitudinal decline in FEV1 as the outcome (and smoking status and 1975 FEV1 as additional predictor variables) revealed s significant (p<.05) association between having an acute cross-shift response in 1975 and subsequent longitudinal change in FEV1. Adjusted mean values for FEV1 decline were -63 ml/yr (se:9.1) for responders and -41 ml/yr (se:4.5) for non-respondere. We conclude that an acute responae to grain dust as measured over one work shift is a significant predictor of an increased risk of subsequent chronic airflow obstruction.

HLA DQB1 AND DPB1 ALLELES ARE ASSOCIATED WITH ISOCYANATE-SENSITIVE OCCUPATIONAL ASTHMA. 1Bignon J, 1Aron A, 2Li Ya Ju, 1Choudat D, 5Pauli G, 6Fabbri L, 4Garnier R, 4Deschamps D, 3Krishnamoorthy R, 1Lockhart A and 1Swierczewski E. 1UFR. Cochin Port-Royal, 2Institut Biomédical Cordeliers, 3U120 Hôpital R. Debré, 4Hôpital F. Widal, Paris, 5Hospices Civils, Strasbourg, France, 6Universita Padova, Italia.

Allergic asthma is useful in evaluating genetic factors that control human immune responsiveness. We investigated whether HLA class II genetic markers were involved in susceptibility or resistance to isocyanate-sensitive asthma. Blood samples were collected in three groups of unrelated Caucasoid and non Caucasoid adults: 1.patients with isocyanate-induced asthma documented by isocyanate inhalation challenge. 2.exposed healthy individuals from the same workplace. 3.healthy controls living in the same area. Genomic DNA was extracted from peripheral blood lymphocytes. The second exon of the HLA DQA1, DQB1 and DPB1 genes which is highly polymorphic, was selectively amplified by the PCR method. Genotyping was carried out by digestion of the amplified DNA products with allele specific restriction endonucleases which can recognize allelic variations in the polymorphic exon. We found no significant differences in the frequency of DQA1 alleles between patients and controls. Allele 2 and allelic combination 2/X of the DQB1 locus were associated with susceptibility to disease. Conversely allele 1.1, allelic combination 1.1/X from DQB1 locus and/or allelic combination 4.1/4.2 of the DPB1 locus confered dominant protection in 64% of the exposed healthy controls (relative risk=0.09). Our results are consistant with the hypothesis that immune mecanisms are involved in isocyanate-induced asthma.

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ABSTRACTS

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