Tu-P-3 INFLAMMATORY BIOMARKERS IN ASPHALT WORKERS

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Background and aims: Asphalt is a mixture of bitumen with crushed stone/gravel. It is put down hot on the road by asphalt pavers, who are exposed to dust, oil mist, polycyclic aromatic hydrocarbons and nitrogen dioxide. In the Nordic countries this kind of work is seasonal, carried out during the summer months. We have recently reported over-season increases of IL-6 and Clara cell protein (CC16) in serum of asphalt pavers (Ulvestad B et al. Scand J Work Environ Health 2007;33:114-122 and Ulvestad et al. J Occup Environ med 2007;49:1073-1078). We have performed additional analyses of an acute phase reactant (Amyloid A) in order to further assess inflammatory effects of such work.

Methods: Blood samples were collected just before (April-May) and in the end of (Sept-Oct) the paving season in 72 asphalt pavers and an internal control group of 51 low-level exposed asphalt plant operators (N=32) or engineers (N=19) from the same company. Serum was analysed for Amyloid A (S-AA) by ELISA (Anogen, Ontario,Canada). The within-subject differences over season were highly skewed (also after log-transformation) and examined by the Wilcoxon rank sum test.

Results: There was a slight but significant group difference with respect to S-AA, with a slight increase (median 0.1 mg/L) in the pavers vs. a slight decrease (median -0.25) in the control group (P=0.046). After exclusion of some outliers a t-test on ln-transformed S-AA levels showed a similar result (P=0.04). The effect was most pronounced in the non-smokers. S-AA levels were significantly correlated to S-CRP and S-IL-6.

Discussion and conclusions: S-AA is a proinflammatory adipokine and an acute phase reactant. S-AA levels seem to predict cardiovascular disease in humans (Ridker PM et al. N Engl J Med 2000;342:836-843), and animal studies indicate that it may also be a risk factor for atherosclerosis (Chait A et al. J Lipid Res 2005;46:389-403). It has been shown to be affected also in experimental exposure to combustion particles (Barregard et al. Inhal Toxicol 2006;18:845-853). S-AA may be a useful indictor in assessment of inflammatory effects of air pollution – in occupational as well as environmental settings.

Tu-P-4 PREDICTORS OF CLARA CELL PROTEIN (CC16) LEVEL: AN INVESTIGATION OF CC16 AS A SILICA EXPOSURE BIOMARKER.

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Background and aims: Clara cell protein (CC16) is a protein that is mainly produced by Clara cells in the respiratory epithelium. CC16 can be measured in bronchoalveolar lavage specimens, serum, urine and sputum. Clara cells and CC16 may play a role in protecting the lung from oxidative stress. A change in CC16 reflects chronic damage to Clara cells and may therefore be a useful biomarker for crystalline silica induced lung damage. This paper presents an analysis of predictors of CC16 in gold miners exposed to silica and unexposed controls.

Methods: 118 African male volunteers participated in this cross-sectional study. They were recruited from a gold mine (silica exposed, n= 64), a blood donor service (silica non-exposed n= 37) and a hospital HIV clinic (silica non-exposed n= 18). Data were collected on age, work history, smoking habits and HIV status. CC16 was assayed in the serum using an ELISA kit. Multiple linear regression was performed with post-regression residuals analysis to identify the factors that might explain the observed variation in CC16.

Results: Arithmetic mean (AM), geometric mean (GM) and geometric standard deviation (GSD) for the whole group CC16 level were 6.0ng/ml, 5.27ng/ml and 1.67ng/ml. AM, GM, and GSD for the silica exposed group were 5.56, 4.87 and 1.69, and for the unexposed group were 6.49, 5.77 and 1.64. The CC16 results were log-normally distributed. T-tests for unpaired data with equal variance indicated a crude relationship between silica exposure and CC16 levels, $p = 0.074$; and smoking and CC16 levels, $p = 0.0001$. Multiple regression with the factors investigated in this study explained 20% of the variation (coefficient of determination $r = 0.19$) and the model identified exposure to silica as a significant risk for a lower level of CC16 ($p = 0.024$) smoking was no longer significant ($p = 0.082$).

Discussion and conclusions: CC16 levels were lowered in silica exposed subjects and were not affected by HIV status. CC16 is a suitable candidate for further research. Factors that further explain the variance in CC16 levels need to be identified before it can be confidently used as a biomarker for silica dust exposure

Tu-P-5 URINARY MERCURY AND BIOMARKERS OF EARLY RENAL DYSFUNCTION IN MERCURY EXPOSED CHLORALKALI WORKERS

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Background and aims: Exposure to elemental mercury can cause a variety of adverse health effects and the kidney is an important target organ. As part of an EU founded project, EMECAP, biomarkers of early