ANALYSIS OF SOME BIOMARKERS TO EVALUATE THE OXIDATIVE STRESS POTENTIALLY INDUCED BY TOLUENE EXPOSURE

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Introduction

Oxidative stress is a condition of unbalance between the pro-oxidant status and the antioxidant protection. Toluene is known as a toxic substance able to cause clinical signs of central nervous system or hepatic and renal changes.

This study evaluates the oxidative stress induced by toluene working exposure, using specific biomarkers.

Methods

We studied the oxidative stress in 187 workers of a firm producing gummed texture exposed to toluene and in a control group of 150 workers of the same firm, not exposed to the toxic substance.

We evaluated environmental and personal exposure levels to toluene and the following oxidative stress biomarkers:

- level of plasmatic hydroperoxides ;
- amount of the total plasmatic antioxidant barrier to determine the capacity of the single patient to control the production of free radicals;
- amount of plasmatic SH groups;
- level of 8hydroxy-2deoxiguanosine, which is a biomarker of DNA damage detectable in urine
- ELISA-measured isoprostanes, producted from oxidation of arachidonic acid, which are considered a reliable marker of oxidative stress.

We also evaluated the same oxidative stress biomarkers in all the workers of the control group.

Results and discussion

The results showed a relevant exposure to toluene, even if under the T.L.V. value, in personal and environmental samples.

Biomarkers of oxidative stress were altered in 67 workers (35.8%) exposed to toluene with a statistically significant difference with the control group ($p<0,01$).

The different markers of oxidative stress didn't show a univocal behaviour in every worker, and this fact forced us to plan in our next researches a screening of the complete set of biomarkers tested in this first study, to avoid false negative results.

We think that, even if our results have to be confirmed in larger and more structured studies, the evaluation of biomarkers of oxidative stress could be a useful research field to investigate a possible precocious work damage from toxic substances.

NON-INVASIVE ASSESSMENT OF CHROMIUM EFFECTS ON RESPIRATORY TRACT IN ELECTROPLATING WORKERS

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Occupational exposure to chromium compounds, which are widely used in industry, may cause airway inflammation and bronchial asthma.

In this study we investigated pulmonary function, chromium in urine and in induced sputum, induced sputum cellularity and markers of inflammation in exhaled breath condensate (EBC) and in nasal lavage fluid of 16 electroplating workers exposed to chromium at lower concentration than current TLV and in 9 non-exposed workers.

All study participants were non smokers without active lung disease. Urinary chromium was 8,64 mg/g creat (SD 6,19).

Lung function values were normal for both groups. Chromium in induced sputum was higher in exposed workers (7.90 mg/L, SD 3.4 vs 1.78 mg/L, SD 0.25). Total leukocyte and neutrophils counts in induced sputum were not significantly higher in exposed subjects (82.98±49.00x104cell/ml vs 68.89±22.71x104cell/ml; 53.08± 34.79x104cell/ml vs 40.45±12.52x104 cell/ml).

In EBC median Nitrite concentration was significantly increased in exposed subjects (4.35 mmol/L, 5°-95° percentile:1.88-10.13 mmol/L vs 0.11 mmol/L, 5°-95° percentile: 0-0.72 mmol/L) ($p<0.001$). IL-6 and TNF-a were not detectable.

Median IL-6 concentration in nasal lavage fluid was higher in exposed workers (5.72 pg/ml, 5°-95° percentile: 0-65.25 pg/ml vs 0.28 pg/ml, 5°-95° percentile: 0-1.7 pg/ml) ($p<0.01$). No differences in Eosinophil Cationic Protein concentration were found. TNF-α was not detectable. In exposed workers urinary chromium levels where not correlated with any marker of inflammation and with chromium in induced sputum.

For the first time this study uses all this 3 non invasive methods to assess early changes in respiratory tract in workers exposed to chromium. These results are suggesting an inflammatory/irritative action of chromium on upper and lower airways which appears not correlated with chromium absorption as indicated by urinary concentration of this metal.

BIOLOGICAL MONITORING OF OCCUPATIONAL EXPOSURE TO STYRENE AND STYRENE-(7,8)-OXIDE

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This study investigated the capability of some urinary and haematic biomarkers to discriminate among different levels of occupational exposure to styrene (Sty) and styrene-(7,8)-oxide (StyOX) and evaluated the influence of smoking habit and genetic polymorphism of metabolic enzymes GSTM1 and GSTT1 on these biomarkers. With this aim, we recruited workers of the reinforced plastic industry (n=8), of the paint and dye industry (n=13), and a group of controls (n=22). Median personal exposure to airborne Sty and StyOX in the different working activities was 14.8, 3.1 and 0.3 mg/m3, and 126, 13 and <5 µg/m3, respectively, as evaluated by repeated measurements. These chemicals were strictly correlated with each other (Pearson r = 0.826), the ratio between Sty and StyOX being about 1000:5. Personal exposure was significantly higher in exposed workers than in controls and, among workers, in subjects of the reinforced plastic industry. Urinary biomarkers, namely unchanged styrene (StyU), mandelic acid (MA), phenylglyoxylic acid (PGA), phenylglycine (PHG), 4-vinylphenol (4-VP), and mercapturic acids (M1 and M2) were higher in end- than in pre-shift samples and significantly correlated with both airborne Sty and StyOX. The best correlations were observed between end-shift MA or MA + PGA and airborne Sty (r = 0.890 or 0.886, respectively). The excretion of mercapturic acids was 6-fold higher in subjects with GSTM1 active genotype in comparison with those with null genotype. Cysteinyl albumin and hemglobin adducts of StyOX could not distinguish the different exposure categories investigated. In conclusion, in both reinforced plastic and paint and dye industry there was co-exposure to airborne Sty and StyOX. Among the different biomarkers urinary MA and PGA and their sum showed the best capability to discriminate different exposures and are recommended for Sty exposure assessment starting from a level of 1 mg/m3.

COMPARATIVE EVALUATION OF URINARY MTBE AND BENZENE AS BIOMARKERS OF EXPOSURE TO URBAN TRAFFIC

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Benzene and methyl tert-butyl ether (MTBE) are found in urban environments as a consequence of autovehicular traffic; in fact both these compounds are added to fuels to increase octane ratings and/or reduce carbon monoxide emissions. Aim of the present study was to evaluate the possibility of using excretion of urinary MTBE (U-MTBE) and benzene (U-BENZ) as biomarkers of exposure to traffic. With this aim 127 Milan urban policemen, working as traffic wardens, were investigated. Spot urine samples were obtained prior to and at the end of the workshift (7.30-13.30 or 13.30-19.30), in different seasons. Analysis was performed by headspace-solid phase microextraction GC-MS. Median levels of airborne benzene was 9.6 µg/m3 (range 4.0-90.2 µg/m3). Urinary levels in the different seasons varied from 74 to 164 ng/L (50-657 ng/L) and from 85 to 277 ng/L (21-5065 ng/L) for U-MTBE and U-BENZ, respectively. U-MTBE increased of about 14% during the workshift, independently from the moment of the shift (morning or afternoon). U-BENZ increased of 27% in the afternoon, but decreased of 15% in the morning. An influence of the different