Human Bitumen Cross-shift Study: Chemical-irritative and Genotoxic Effects of Fumes and Aerosols of Bitumen under High Processing Temperatures on the Airways

M. RAULF-HEIMSOTH, K. SCHOTT², H.J. SCHICKER², H.P.RIHS¹, B. MARCZYNSKI¹, G. ZOUBEK², J. ANGERER³, B. PESCH¹, R. RUMLER², R. RÜHL⁴, R. MERGET¹, T. BRÜNING¹

¹Berufsgenossenschaftliches Forschungsinstitut für Arbeitsmedizin (BGFA), Institut der Ruhr-Universität Bochum, Germany

²Tiefbau-Berufsgenossenschaft, Germany

³Institut und Poliklinik für Arbeits-, Sozial- und Umweltmedizin (IPASUM), Universität Erlangen, Germany

⁴Bau-Berufsgenossenschaft Frankfurt/Main, Germany

Introduction

Possible hazards of fumes and aerosols from bitumen have been a subject of discussion for several years. In 2001, the Committee for hazardous substances (AGS) reduced the threshold limit value for fumes and aerosols of bitumen to 10 mg/m³. For mastic asphalt workers, who are exposed to clearly higher concentrations, the limit value was temporary deferred. These workers are currently undergoing selective occupational health monitoring [1]. Since 1977 bitumen is also listed as a suspected carcinogen in the list of German MAK and BAT values [2]. Evidence for genotoxicity and carcinogenicity was mainly obtained from animal studies and in vitro assays, which indicated that asphalt fumes are genotoxic and produce local skin carcinomas in mice after dermal exposure. Further, bitumen and asphalt fumes are also related to irritation in the respiratory tract (adverse effects) and possibly related to chronic bronchitis (non-adverse effects). The finally effect of bitumen fumes and aerosols in human, if chemical-irritative or genotoxic, is being so far controversial discussed.

In 2000 we started a project focused on a comprehensive examination of a group of substantially exposed mastic asphalt workers to determine possible chemical-irritative and genotoxic effects especially on the airways induced by exposure to fumes and aerosols of bitumen.

Here we present the first results of the pilot study comprising biological monitoring data as well as analyses of lung function parameters and sputum.

Collective and methods

In total 95 workers (all insured by the Tiefbau-BG) were examined before and after a work shift. It concerns 46 workers, who were exposed to fumes and aerosols of bitumen under high processing temperatures at indoor workplaces and 49 workers with a comparable job profile but without exposure to bitumen (controls). A detailed structured questionnaire related to work activities and diseases was used. Before (pre-shift) and after work (post-shift) lung function parameters of the workers flanked the examination. At both time points, the collection of nasal lavage fluids, sputum and blood samples allowed the determination of a possible airway inflammation of the lower and upper airways as well as genotoxic effects induced by components of bitumen fumes and aerosols. In addition to the analysis of PAH metabolites in urine samples collected pre- and post shift, personal- and environmental-related exposure measurements (ambient-monitoring) were also performed.

Results

39 out of the 46 exposed workers handled with bitumen in new building subterranean garages and were exposed to bitumen fumes and aerosols under high processing temperatures, whereas further seven persons worked also indoors, but at a restoration area. Due to a not comparable exposure situation, this seven workers were analyzed separately. The mean age of the bitumen exposed workers as well as the not exposed controls was 37 years. Regarding smoking behaviour a significant difference existed between exposed and non-exposed control workers (p<0.01). Since smoking constitutes a confounder for the further examination, four groups were provided for the analysis:

- exposed non smokers (n = 11),
- exposed smokers (n= 28)
- not exposed non smokers (n= 29) and
- not exposed smokers (n= 20).

The further examinations were performed according this group classification in pre/post-shiftcomparisons. The first step of the data analysis contained a descriptive analysis of the biomarker under consideration of the exposure, the smoking status and the shift course.

The first evaluations of the PAH-metabolites in urine showed for the sum of the hydroxyphenanthrene as well as for the single hydroxyphenanthrenes (both referring to the

creatinine value) a clear significant difference between the bitumen-exposed and the bitumen-non exposed worker. In the exposed group the post-shift values were significantly increased (p=0.0001). Also the post/pre-shift ratio was significantly influenced by the exposure (p=0.0002). Smoking affected also the elimination of hydroxyphenanthrene (p=0.003 for the post-shift values). No smoking effect was visible, when the post/pre-shift values were calculated (p=0.53). Furthermore, the data demonstrated that the pre-shift hydroxyphenanthrene values in the exposed smoker group were higher when compared with the values of the bitumen-exposed non smokers. It became apparent, that the exposure has a synergistic influence on the elimination of these PAH-metabolites.

The analysed lung function parameters (among other parameters FEV_1 , FEV_1 % predicted, FVC and FVC% predicted, MEF, PEF etc.) revealed a significant influence of the exposure for the FEV₁% predicted value (post/pre ratio: p=0.03). In summary, it can retain for the lung function that for the exposed group the slightly higher pre-shift FEV₁% predicted values were determined (but not significant). If there is a so called "healthy worker effect", this effect can not be evaluated, yet. Post-shift values of the exposed group were slightly lower. The determined post/pre-ratio was significantly influenced by the exposure (p=0.03). The smoking behaviour had no significant influence on the lung function parameters at the "cross-shift"-examination. The changes of the lung function parameters give a hint that a restrictive ventilation disorder may be induced by bitumen fume and aerosol exposure.

Since the collection of nasal lavage fluids as well as of induced sputum is not invasive, several repeated collection steps for the test persons are less cumbering and technically possible [3]. Therefore, next to blood and urine these samples could also be collected pre- and post-shift. Using these samples from the upper and lower respiration tract collected before and after work shift cell population will be determined by differential cytology and a quantification of soluble inflammation mediators (ECP, IL-8, IL-5, TNF α , albumin, NO, IL-1 β) will be undertaken. For example, the IL-8 concentration in sputum (quantification by specific commercial ELISA) of the post-shift samples showed a clear relation to the exposure as well as to the smoking status, but seemed to be age independent. The IL-8 post/pre-ratio values reflected itself likewise another– but lower – exposure influence. In the pre- as well as in the post-shift sputum samples a significant smoking factor was visible, again controlled by the formation of the post/pre-ratio.

Conclusions

The present preliminary results of our pilot study showed an increase of the elimination of PAHmetabolites in the post-shift urine samples and acute effects on the airways induced by fumes and aerosols of bitumen. Furthermore, our data clearly demonstrated that it will be necessary to amplify the epidemiological power of this study by expansion of the collective and stratifying them concerning smoking behaviour. On the other hand an amplification of the examination of genotoxic effects with 'state of the art' molecular biological methods will be necessary to get sophisticated results.

Literature

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