Biological monitoring of environmental exposure to PAHs in the vicinity of a Söderberg aluminium reduction plant

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Abstract
Objectives—To assess environmental exposure to polycyclic aromatic hydrocarbons (PAHs) in the vicinity of a Söderberg aluminium reduction plant in Shawinigan, Canada with urinary 1-hydroxypyrene (1-OHP) as a biomarker.

Methods—Urine samples were collected from 20 non-occupationally exposed subjects living less than 500 m from the plant and from 20 controls living in Trois-Rivières, another industrial town 40 km from Shawinigan. Concentrations of 1-OHP were measured by high-performance liquid chromatography (HPLC).

Results—Among controls, geometric mean (range) 1-OHP concentrations were 0.046 (0.012–0.116) μmol/mol creatinine in non-smokers and 0.125 (0.051–0.282) μmol/mol creatinine in smokers. Among exposed subjects, values were 0.103 (0.056–0.196) μmol/mol creatinine in non-smokers and 0.250 (0.112–0.448) μmol/mol creatinine in smokers. Excretion of 1-OHP was significantly higher in exposed subjects than in controls among non-smokers and smokers (P<0.05).

Conclusion—Based on urinary 1-OHP as a biomarker, it seems that living near an industrial point source of PAHs is associated with higher exposure. The health significance of this finding will require further investigation.

Keywords: biological monitoring; polycyclic aromatic hydrocarbons; environmental pollution

Aluminium reduction by the Söderberg electrolysis process is known to release large amounts of carcinogenic polycyclic aromatic hydrocarbons (PAHs) such as benzo[a]pyrene. An increased rate of lung cancer was found among Söderberg plant workers exposed to coal tar pitch volatiles containing PAH, and the risk of mortality from lung cancer was shown to increase with cumulative exposure. In Canada, high concentrations of carcinogenic PAHs were measured in areas surrounding Söderberg plants in 1989–91: mean benzo[a]pyrene concentrations in Jonquière and Shawinigan (two towns where Söderberg plants are operating) were of 36 and 28 ng/m³, whereas in Montréal these values were 0.6 ng/m³ downtown and 0.8 ng/m³ just beside a main road. But little is known about the potential effects of industrial emissions of PAHs on the health of neighbouring human populations. There is a need for further research in these areas, and accurate assessment of exposure to PAHs is a necessary step in such investigations.

Urinary 1-hydroxypyrene (1-OHP) is used as a biological marker of occupational exposure to PAHs in several work environments. It was also used recently for monitoring of environmental exposure, and was significantly increased in an area heavily polluted by industrial PAH emissions and by burning of black coal, compared with a non-industrialised area.

This pilot study was undertaken to investigate environmental exposure to PAHs of people inhabiting the vicinity of an aluminium reduction plant that used the Söderberg process in Shawinigan, Canada, with urinary 1-OHP to monitor this exposure.

Material and methods
The exposed group was recruited from electoral lists, among people living < 500 m from the aluminium plant of Shawinigan. In that particular residential area, the arithmetic mean concentration of benzo[a]pyrene in ambient air was 16 ng/m³ in 1993–4. (M Bisson and P Walsh, Québec Ministry of Environment and Wildlife, unpublished data quoted with permission).

Controls were residents of Trois-Rivières, a town 40 km from Shawinigan. Trois-Rivières is industrialised, but has no major industrial source of PAHs. Control group A consisted of residents of Saint-Philippe, an area of Trois-Rivières which is close to a port, a paper plant, and the trans-Canada highway. These subjects were identified on electoral lists and recruited by the same procedure used for the exposed group. Control group B consisted of Public
Health Department employees and the husbands of two of them.

All subjects were aged between 30 and 45. Their smoking status, employment history, and medicinal use of products containing tar for the past six months were verified. Those with occupational or medicinal exposure were excluded: a roofer handling tar, a dustman working behind a diesel lorry, and an aluminium plant worker were excluded from the exposed group and three people who used lotions containing tar for treatment of psoriasis were excluded from control group B. Active smokers were included in the study, but considered separately for statistical analysis. Passive smokers (non-smokers living with a smoker or working in a smoke polluted environment) were excluded. Finally, 13 subjects were included in control group A, seven in control group B, and 20 in the exposed group.

Morning urine samples (before subjects left their home to work) were collected between 12 and 20 June 1996. They were collected over thymol, kept in ice for one to five hours (while other samples were collected), and then frozen and kept at −20°C until chemical analysis.

The 1-OHP concentration in urine samples was measured by the reversed phase high performance liquid chromatography (HPLC) fluorescence method of Jongeneelen et al modified by Bouchard et al.7 The HPLC system consisted of a model AS-100 HPLC automatic sampling system (Bio-Rad, Richmond, CA, USA), a model 250 binary pump (Perkin-Elmer, Buckingham, England), a 250 × 4.6 mm Supelcosil LC18 column (Supelco, Oakville, Ontario, Canada), and a Perkin-Elmer LS-40 fluorescence detector. Excitation and emission wavelengths were set to 242 and 388 nm, respectively.

Study groups were compared, with SPSS 6.1 statistical software, by the rank comparison U test of Mann-Whitney (two tailed). The U test was preferred to parametric tests because of the small size of study groups. Differences were considered significant at \( P < 0.05 \).

**Results**

Among non-smokers, geometric mean concentrations of 1-OHP were 0.057 μmol/mol creatinine in control group A, 0.038 μmol/mol creatinine in control group B, and 0.103 μmol/mol creatinine in the exposed group. Among smokers, mean values were 0.125 μmol/mol in control group A and 0.250 μmol/mol creatinine in the exposed group (figure). There were no smokers in control group B. As non-smokers from controls A and B were not significantly different \( (P=0.3173) \), they were combined for subsequent statistical analysis. Urinary 1-OHP was significantly higher in exposed subjects than in controls, both among non-smokers \( (P=0.0026) \) and among smokers \( (P=0.0300) \).

Urinary 1-OHP was also twofold higher in smokers than in non-smokers. This increment was significant in exposed subjects \( (P=0.0016) \) and in controls \( (P=0.0126) \).

**Discussion**

This study showed that subjects living near the aluminium reduction plant of Shawinigan have a higher urinary concentration of 1-OHP than subjects living in another industrialised town with no such comparable point source of PAHs; it is interesting to note that mean 1-OHP concentration in exposed non-smokers was almost as high as in control smokers. These results suggest that higher environmental PAH contamination is associated with significantly higher internal exposure to PAHs.

On the other hand, 1-OHP concentrations measured in the present study were far below concentrations found at the end of the shift by Jongeneelen et al for Söderberg plant workers: in the low exposure group (electrolysis and foundry workers) mean values were 0.47 μmol/mol creatinine for non-smokers and 0.74 for smokers; and in the high exposure group (bake oven, anode factory, and pot relining workers), mean values were 3.84 μmol/mol creatinine for non-smokers and 7.19 μmol/mol creatinine for smokers.8 Obviously, environmental exposure to PAHs which seems to occur in the vicinity of Söderberg plants is much lower than the exposure of Söderberg plant workers.

This study was not designed to find by which particular route subjects were exposed by PAHs. Both inhalation and dermal route are known to contribute to occupational exposure to PAHs, but non-occupational exposures have been less studied and the relative contribution of exposure routes is unknown.

Exclusion of people occupationally or otherwise exposed to PAHs, and sample collection in the morning (after subjects spent the past 10–12 hours at home) reduced the influence of exposure from other sources than the environment of subjects’ home area. But this influence may not have been entirely eliminated, as the biological half life of 1-OHP is about 12 hours.9 Environmental sources other than industrial emission are unlikely to have significantly influenced the results. Sampling took place in June, when houses are not heated, so there is no domestic wood burning. Petrol and
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diesel engines also release PAHs into the environment, but as already mentioned, PAH contamination of outdoor air by traffic is much lower than contamination by industrial emissions. Furthermore, control group A had potentially the greatest exposure to PAHs released by engines: all the members of that group lived less than 1 km from the trans-Canada highway, and no such traffic artery crosses Shawinigan.

Consumption of grilled or smoked foods is also a source of exposure to PAHs and a determinant of 1-OHP excretion. It is therefore a potential confounding factor and should be carefully assessed in further investigations. A food diary filled in by subjects before urine sampling could help to estimate dietary intake of PAHs.

The increase of 1-OHP excretion in smokers is not surprising, as this biomarker has been shown to be correlated with daily cigarette consumption in subjects with no occupational exposure to PAHs. Our values for control non-smokers and smokers are close to values previously published by this laboratory for control subjects (0.07 and 0.12 μmol/mol creatinine in non-smokers and smokers). These findings confirm the necessity to constitute separate groups of non-smokers and smokers when measuring 1-OHP for environmental exposure studies.

In conclusion, urinary 1-hydroxypyrene used as a biomarker of environmental contamination by PAHs suggests that emissions by industrial point sources result in an increased exposure of neighbouring populations. No direct relation has been established between urinary 1-OHP and risk of lung cancer, so 1-OHP cannot yet be used for a quantitative risk assessment. However, because of the genotoxic and carcinogenic potencies of some PAHs, a significant increase of exposure to PAHs in a human population is by itself information of interest that may warrant further investigation.

We are grateful to the 40 volunteers from Shawinigan and Trois-Rivières who participated in this study and to Micheline Pelletier for 1-OHP analyses. This research was supported by the Regional Health and Social Services Board of Mauritie-Bois-Francs.


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*Occup Environ Med* 1997 54: 619-621
doi: 10.1136/oem.54.8.619

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