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1-Hydroxypyrene Levels In Coal-Handling Workers at a Coke Oven

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Abstract

An environmental and medical survey was conducted at the coal-handling area of a coke oven, where workers came in contact with coal-tar sludge. The purpose of the study was to determine if skin contact with coal-tar sludge was an important route of exposure to pyrene because workers were observed to have substantial contact with the sludge. Environmental monitoring revealed minimal airborne exposure to pyrene, a byproduct of the coke distillation process; only one personal breathing zone sample detected pyrene, and at a level of 0.001 mg/m^3. However, the mean preshift urinary 1-hydroxypyrene concentration was 1.00 μmol/mol creatinine (range, 0.16 to 2.96 μmol/mol creatinine) and the mean postshift level was 1.7 μmol/mol creatinine (range, 0.24 to 4.85 μmol/mol creatinine) ($P < 0.01$). These levels probably reflect absorption as a result of skin exposure.

The mixing of coal-tar sludge with coal before being processed in coke ovens is a method to minimize the amount of hazardous waste requiring disposal. The National Institute for Occupational Safety and Health (NIOSH) received an employee request for a health hazard evaluation at a coke oven because of worker concern about exposure to this sludge during both the mixing process and when the coal-sludge mixture is brought to the coke oven for burning. Based on observations of work practices and worker interviews, it became apparent that there was potential for substantial worker exposure to coal-tar sludge from skin contact and that air sampling alone would miss this route of exposure.[1] Biologic monitoring for 1-hydroxypyrene (1-HP), a metabolite of pyrene, was done to more accurately assess worker dose.

1-HP is the major metabolite of pyrene, a compound found in coal tar, and is formed by the liver and excreted in urine and stool.[2] The pyrene to total polycyclic aromatic hydrocarbon (PAH) ratio is relatively constant in the coke-oven environment, and monitoring of urinary 1-HP is a viable method of assessing total PAH exposure.[3] Both environmental monitoring and biologic monitoring were performed during this study. However, this article will concentrate on the 1-HP analysis. A comprehensive discussion of the evaluation methods and environmental monitoring results are presented in the health hazard evaluation report.[4]

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Approximately 400 employees work at this facility, which can produce about 1,000,000 tons of coke annually at full capacity. Metallurgical coke is the residue of devolatilized bituminous coal, and is used as a fuel and reductant in blast furnaces for steel-making. This process entails heating coal to temperatures of 900 to 1100°C in the absence of oxygen, to distill out tars (including coal-tar pitch volatiles [CTPVs], polycyclic aromatic hydrocarbons [PAHs}, light oils (including benzene and toluene), ammonia, water, sulfur compounds, and a gaseous byproduct referred to as coke-oven gas, to produce a substance with a substantially higher carbon content.[5] Coke-oven gases consist of 75 to 85% hydrogen and methane and are used as a plant fuel.

Coke-oven emissions and byproducts contain both known (benzene) and suspected (PAHs) carcinogens.[6] Bladder, lung, and kidney cancer have been linked to chemical exposures of coke-oven workers.[7] Redmond et al found a relative risk for kidney cancer of 7.5 in coke-oven workers.[8] A study of aluminum workers exposed to CTPVs reported an excess of cancer of the lung, stomach, esophagus, and bladder.[9]

Slot-type coke ovens arranged in batteries (a series of ovens), which are capable of chemical (byproduct) recovery, were used at the facility. At this coke plant, a wet load (charge) of approximately 28 tons of coal yields approximately 20 tons of coke per cycle. A cycle will take from 16 to 24 hours to complete. The resulting coke is then pushed out of the oven and quenched with water. Standpipes on each oven collect the gases generated from the coal during the carbonization process. These standpipes are connected to large collecting mains, which transport the effluent to the byproducts plant, where the gases are separated and processed. The coal-handling building, consisting of large storage bins containing various grades of coal, is adjacent the byproducts plant.

The blending of coal-tar sludge with coal has been an ongoing process at this coke plant since 1991. Coal-tar sludge, or tar decanter sludge, is the residue remaining from the byproducts recovery process. It is composed of small coal particles (33%) and coal tar (67%). The sludge is obtained on a daily basis from four hot-tar drain-tank decanters that discharge the sludge into steam-heated sleds, which are moved by truck to the blending area, an open concrete-lined pit used to store the sludge. The blending process consists of using a front-loader to mix sludge with coal at a 1:10 ratio. (During the colder winter months, the sludge is too viscous to work, so the process is shut down.) A screw conveyor located in this area is used to mix and convey the sludge-laden coal into the appropriate storage pile. Sludge-laden coal is then transported to a location near the coal-handlers building (known as the shaker area) where it is dispensed through a grate into a collection pit and then on to a conveyor system. The conveyor transports the sludge-laden coal to the coal-handlers building, where it is delivered to a storage bin. Of the final coal blend delivered to the coke ovens, approximately 3% is the sludge-laden coal.

Three employees reported that exposure to coal-tar emissions in an area known as the #1 hole was particularly intense, especially in the summer. The "hole" is an underground area that surrounds the conveyor belt that transports coal, including coal that has been mixed with coal tar or sludge, to the storage hoppers. It is open only at one end and is accessible by a stairway, and there is no forced ventilation. Workers reported that coal-tar emissions concentrate in this area and that skin contact with the sludge was likely, particularly during cleaning and maintenance operations.

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Environmental Monitoring

During the survey, both personal breathing zone (PBZ) and bulk samples were taken at the work site. Ten employees were selected for environmental monitoring based on the materials they handled, work practices, and job duties. CTPVs were detected on filters from seven of ten (70%) PBZ samples, and detectable PAHs were measured on three of the filters. Lower-molecular-weight PAHs were detected on all ten backup sorbent tubes from the PBZ samples. The highest CTPV concentration (0.35 mg per cubic meter [mg/m^3]) and the greatest number of PAHs were found on a PBZ sample obtained from a day-shift laborer. Naphthalene was detected on all samples (both area and PBZ), and the highest PBZ concentration (3.5 mg/m^3) was measured in a laborer operating the coal-sludge blending auger. Only a trace of pyrene was detected on one personal sample, at a level of approximately 0.001 mg/m.[3] Both detectable and quantifiable levels of benzene were found on five of the ten PBZ samples and in both area samples. A concentration of 0.8 parts per million (ppm) benzene was found on an 81-minute area sample collected in the cab of a vehicle loading the coal-sludge mixture. All coal sludge bulk samples contained numerous common PAHs associated with coal-tar products, including the five PAHs listed in the Occupational Safety and Health Administration (OSHA) definition of CTPVs. Pyrene was found in the analysis of the bulk samples of coal-tar sludge at levels of between 6.3 and 36 mg/gm.

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Methods

All workers in the coal-tar sludge-handling area on the day of the survey (June 1994) were eligible to participate in the 1-HP analysis and included all of the workers who had participated in the PBZ environmental monitoring. On the days of the evaluation, there were 22 workers eligible for inclusion in the study. Participants were asked to provide a urine sample and to complete a questionnaire that asked about work practices, diet, and smoking, and were required to complete a Human Subjects Review Board-approved consent form before participating.

Workers were instructed to report to the nurses' station upon arrival at work and before leaving so that pre- and postshift urine samples could be collected to determine if there was an increase in 1-HP during the course of the workday, and for fitting of the environmental monitors. The urine samples were frozen after collection and returned to Cincinnati for analysis. Analysis was performed by the method described by Tolos et al using high-performance liquid chromatography with a fluorescence detector.[10]

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Results

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1-Hydroxypyrene in Urine

Urine samples were collected from 18 of the 22 eligible workers from all work shifts. Job classifications of the workers included: (1) coal-handler operators (six workers), (2) coal handler or pump maintenance (five workers), (3) laborers (two workers), (4) other (five workers, including one welder, crane operator, repairman, foreman, and loader operator). All workers were men, with a mean age of 41 years; nine smoked cigarettes.

Preshift urinary 1-HP concentrations ranged from 0.16 to 3.0 µmol/mol creatinine (mean, 1.00) and postshift levels ranged from 0.24 to 4.85 (mean, 1.7). Using a paired t test, this difference was statistically significant ($P = 0.01$). The highest postshift mean 1-HP levels were found in the laborers (3.7 µmol/mol creatinine) (Table 1). Unfortunately, the one worker whose environmental monitor revealed airborne exposure to pyrene was unable to produce a preshift urine sample, and was not one of the 18 workers evaluated in the 1-HP analysis.

Table 1

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Employee Questionnaire

Workers reported that respirators were worn "always" by seven of the workers and "sometimes" by another seven workers. Nine of those 14 (64%) reported on the questionnaire that they were able to smell chemicals while wearing the respirator. However, workers who reported that they smelled chemicals while wearing a respirator had neither a higher preshift 1-HP level ($P = 0.92$) nor a higher postshift 1-HP level ($P = 0.94$) on the day of the survey when compared with workers who did not smell chemicals. NIOSH investigators did not observe any workers wearing respirators in the coal-handling area during either site visit.

Smoking was not found to be related to 1-HP levels. The nine workers who smoked cigarettes had statistically insignificant increased mean preshift levels of 1-HP when compared with nonsmokers (mean, 1.2 µmol/mol creatinine for the smokers as compared with a mean of 0.7 µmol/mol creatinine for the nonsmokers; $P = 0.21$). Postshift levels of 1-HP were similar for both groups (mean, 1.8 µmol/mol creatinine for the smokers and 1.7 µmol/mol creatinine for the nonsmokers; $P = 0.87$).

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Discussion

PAHs may enter the body through different routes, namely, the skin, respiratory system, and gastrointestinal tract.[11] The uptake of PAH through the skin is highly relevant in terms of the internal dose, and dermal absorption is reported to be 50 to 90% of the total PAH uptake among some occupationally exposed workers.[12] Therefore, air monitoring alone may underestimate total exposure of workers, whereas the use of 1-HP as a biological marker for exposure would reflect total exposure. Other advantages of the use of 1-HP as a marker of exposure are that sample contamination is not likely, because 1-HP is a metabolite formed in the body and has a low limit of detection (1.37 nmol/L).[13]

Jongeneelen et al suggested a biologic exposure limit (BEL) of 2.3 μmol/mol creatinine, based on the ACGIH TLV of 0.2 mg/m^3 for CT-PVs.[1] However, the NIOSH REL (full-shift time-weighted average) for CTPVs is 0.1 mg/m^3 of the benzene (or cyclohexane) extractable fraction of the sample and is based on the potential risk of lung and skin cancer. The NIOSH REL would probably correspond to a lower urinary 1-HP concentration. In Jongeneelen's study, the average pre-work 1-HP concentration in urine was 0.91 μmol/mol creatinine.[3] Another study determined that the upper limit of the normal (without occupational exposure) value was 1.31 μmol/mol creatinine.[14] In this study, seven workers (32%) had a postshift 1-HP concentration above the proposed BEL of 2.3 μmol/mol creatinine, and two workers (9%) had a preshift level above that amount.

The effect of smoking on urinary 1-HP excretion among workers exposed to CTPVs is controversial. Jongeneelen et al found that smoking resulted in a greater increase in urinary 1-HP in coke-oven workers than nonexposed (to coke-oven emissions) control subjects.[2] Using thiocyanate excretion as a marker for cigarette smoking, Buchet et al found that smoking can explain only 2.3% of the variance in urinary 1-HP excretion among coke-oven workers.[15] In this study, a greater difference between mean pre- and postshift 1-HP concentrations was found in smokers (0.9 μmol/mol creatinine) than in nonsmokers (0.6 μmol/mol creatinine). Besides the contribution of cigarette smoke to pyrene exposure, it is possible that contamination of the cigarette with coal tar from worker's hands might occur, possibly resulting in both ingestion and inhalation. Another factor that may affect 1-HP levels is treatment for psoriasis with a coal-tar-based ointment, which can result in increased 1-HP excretion; one study found urinary levels of 1-HP of 547 μmol/mol creatinine in coal-tar-treated psoriasis patients as opposed to 0.14 μmol/mol creatinine in untreated patients.[16] In the study presented here, no participants responded that they were using a coal-tar-based ointment.

Studies of other coke-oven workers exposed to pyrene have yielded results similar to those found in this study. In nonsmokers, Zhao et al found a preshift mean 1-HP level of 0.77 μmol/mol creatinine and a post-shift mean level of 1.78 μmol/mol creatinine.[2] However, the workers in that study had higher airborne exposures to PAHs than those monitored during this evaluation. Coke-oven workers in the study by Jongeneelen et al had average airborne exposure to PAHs ranging from 6.9 to 17.0 μg/m^3 and airborne mean pyrene exposure of 0.6 to 2.0 μg/m^3. In the study presented here, pyrene was detected on only one personal sample at a level of approximately 1 μg/m^3. The failure to find airborne pyrene is probably because workers in the NIOSH study were not exposed to coke-oven gases but were predominantly exposed to coal-tar sludge. When the sludge is cool, as it was when handled by the workers in the study presented here, the vapor pressure of pyrene (6.85×10^{-7} mm Hg at 20°C) is low enough to preclude a large airborne exposure.

The results of this study demonstrate the absorption of pyrene in a group of coke-oven workers without measurable pyrene in personal air samples of study participants. For these workers, substantial contact of the skin to coal tar was observed based on work-practice assessments, and absorption was demonstrated through biological measurement of a pyrene metabolite. This exposure was reflected in the increase in urinary 1-HP levels from preshift to postshift samples.

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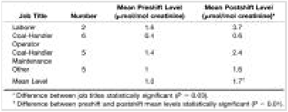

Job Title	Number	Mean Preshift Level (μmol/mol creatinine)	Mean Postshift Level (μmol/mol creatinine)*
Laborer	2	1.6	3.7
Coal-Handler Operator	6	0.4	0.6
Coal-Handler Maintenance	5	1.4	2.4
Other	6	1	1.6
Mean Level		1.0	1.7†

* Difference between job titles statistically significant (P = 0.03).
† Difference between preshift and postshift mean levels statistically significant (P < 0.01).

☐ Table 1

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