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Short Communications

Occupational polycyclic aromatic hydrocarbon exposure assessment by job task in aluminum workers: a biomonitoring study

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ABSTRACT

Introduction: Occupational exposure to polycyclic aromatic hydrocarbons (PAHs) in the aluminum smelting industry poses a significant health risk.

Methods: The study was conducted in 2021 at an aluminum production facility in Sohar Aluminum, Oman, to assess task-specific (cleaning, electrical, mechanical, and operation) occupational exposure to PAHS from the paste plant department. The non-exposed workers at the medical center are chosen as the control group. Personal air samples were collected using a 13-mm glass fiber filter (0.3 µm, SKC Inc.) housed in a Millipore Swinnase cassette. Post-shift urine samples were obtained across task groups during 8-hour shifts. PAHs and urinary 1-OHP levels were quantified through high-performance liquid chromatography (HPLC).

Results: Airborne PAH concentrations ranged from 0.0 to 417.4 µg/m^3. The mean urinary 1-OHP concentration was 7.46 µmol/mol creatinine (range: 0.0–40.9). Electrical and mechanical maintenance workers showed the highest urinary 1-OHP levels. A strong positive correlation existed between airborne PAH and urinary 1-OHP ($r = 0.900$, $p < 0.001$).

Conclusion: Elevated PAH exposure was task-specific, with the highest levels in electrical and mechanical roles. Urinary 1-OHP proved effective for monitoring internal PAH exposure, underscoring the need for targeted exposure reduction strategies.

Keywords: Job task, Occupational exposure, Polycyclic aromatic hydrocarbon, Urinary 1-Hydroxy pyrene

Introduction

Polycyclic aromatic hydrocarbons (PAHs) are persistent organic pollutants with varying structures and toxicities, posing significant human health risks and making them critical for environmental health assessments.[1] The aluminum manufacturing industry represents a primary PAH source through incomplete combustion of organic materials during production processes, creating substantial health risks for workers, including respiratory problems and increased cancer risk from long-term exposure.[1] Biological monitoring using urinary metabolites, particularly 1-hydroxypyrene (1-OHP), has emerged as a reliable method for assessing individual PAH exposure levels and informing risk mitigation strategies.[2]

Although existing research has documented PAH exposure in aluminum production facilities, critical knowledge gaps remain regarding exposure variability across different job roles and work shifts, as well as temporal exposure patterns

that could inform optimal protective strategies.[3] The complex interplay of industrial processes, environmental conditions, and work practices creates intricate exposure patterns that require a comprehensive assessment.[4] However, the aluminum industry lacks detailed studies examining both task-specific and shift-related variations in PAH exposure. This study quantifies urinary 1-hydroxypyrene and associated air PAH concentrations across job classifications and shifts to characterize exposure patterns and establish an empirical foundation for industrial hygienists to develop evidence-based occupational health and safety interventions that address task-specific exposure profiles.

Methods

This cross-sectional study assessed occupational PAH exposure among workers at an aluminum production facility in Oman, which was selected for its use of bituminous coke and pitch in anode production, a known source of PAH emissions. This study was conducted in accordance with the ethical principles of the Helsinki Declaration. The Institutional Ethics Committee approved the study protocol, and all participants provided written informed consent. Thirty-eight male workers, aged 23-52 years (mean, 36.9 years), with at least 2 years of employment, participated voluntarily.

Participants were recruited from Similar Exposure Groups (SEGs) based on job roles in the carbon department's paste plant, including cleaning, electrical, and mechanical tasks. A reference group of non-exposed workers from the port and medical facilities provided baseline comparisons.

Early-morning urine voids were collected from workers and maintained in a cold chain during sample transport. Likewise, the air sampling was conducted utilizing SKC air samplers (226-30, SKC Inc.) that were outfitted with glass fiber filters and XAD-2 adsorption tubes, maintained at a steady flow rate (0.2 L/min). Urine and air samples were collected from March to May 2021 across designated work areas within the aluminum facility. The samples were shipped in dry ice to an AIHA-accredited laboratory for further analysis as per standard protocol. Workplace PAH concentrations were compared against the Deutsche Forschungsgemeinschaft technical reference value of 2 μg/m³ for benzo(a)pyrene (8-hour time-weighted average) for coke oven operations.[5]

Urine samples were collected from each worker before and after shifts at the beginning of the workweek to minimize carryover effects from previous exposures and stored at 4°C until analysis. Urinary 1-hydroxypyrene (1-OHP), a validated PAH exposure biomarker, was analyzed using standard extraction protocols and high-performance liquid chromatography (HPLC) separation with fluorescence detection (LOD: 0.05 μg/L; LOQ: 0.03 μg/L). Analytical quality was ensured through participation in external quality assessment programs (G-EQUAS Erlangen and OSEQAS, conducted twice a year). Creatinine concentrations were measured spectrophotometrically using a semi-automatic biochemistry analyzer to normalize 1-OHP levels and correct for urine dilution[7]. Samples with creatinine concentrations outside the range of 4-34 mmol/L were excluded to ensure reliability.[8]

Exposure metrics (air PAH concentrations and urinary 1-OHP levels) were averaged for each worker and task group, and both measures were log-transformed to achieve normal distributions. Linear associations between airborne PAHs and biological exposure indicators were examined using Pearson correlation analysis and visualized using scatterplots with fitted linear regression lines. Statistical significance was set at $p < 0.05$, and all analyses were conducted using R software version 4.3.2.

Results

Table 1 illustrates significant differences in personal PAH (EPA 16) concentrations among workers by task. The ambient PAH level in the smelter's paste plant was 322 (μg/m³). The occupational exposure assessment revealed substantial variation in PAH concentrations across job categories. Electrical workers showed the highest PAH exposure (mean 85.25 μg/m³), exceeding the German TRK limit of 2 μg/m³ by 42-fold, while mechanical workers had significantly elevated exposure (mean 50.03 μg/m³) at 25 times the regulatory threshold ($P<0.05$), and even cleaning personnel demonstrated concentrations (mean 5.54 μg/m³) that were 2.8-fold above the limit ($P<0.05$). In contrast, D&R technicians and control group subjects exhibited negligible PAH concentrations, which were statistically indistinguishable, indicating minimal or no occupational exposure in these categories.

Table 1: Task-based occupational exposure to concentration of PAHs in air (μg/m³) and associated urinary 1-hydroxy pyrene biomarker levels (μmol/mol Creatinine) among Aluminum industry workers.

	n*	Mean	Median	Range (Min-Max)	Coefficient of Variation (%)	Confidence Interval of the mean	Area PAH (μg/m³)
Task 1: Control							
PAH* in air (μg/m³)	3	0.01	0.00	0.00-0.02	114.5	0.04	ND†
1-OHP in urine (μmol/mol. Creatinine)	3	0.09	0.07	0.05-0.16	51.2	0.1	
Task 2: Cleaning							322.9
PAH* in Air (μg/m³)	2	5.54	5.54	4.76-6.30	19.7	9.8	
1-OHP in Urine (μmol/mol. Creatinine)	2	10.77	10.77	8.89-12.60	60.5	23.6	
Task 3: Electrical (EM-technician)							
PAH* in Air (μg/m³)	2	85.25	85.25	0.80-169.69	140.1	1072.9	
1-OHP in Urine (μmol/mol. Creatinine)	2	20.50	20.50	2.13-38.88	144	233.5	
Task 4: Mechanical (MM Technician)							
PAH* in Air (μg/m³)	12	50.03	1.65	0.02-417.37	220.7	77.6	
1-OHP in Urine (μmol/mol. Creatinine)	12	10.59	6.27	0.80-40.90	104.9	7.4	
Task 5: Others							9.3
PAH* in Air (μg/m³)	7	0.77	0.10	0.0-2.78	0.14	1.03	
1-OHP in Urine (μmol/mol. Creatinine)	7	2.43	2.04	0.04-4.80	105.2	1.6	
Task 6: D&R technician							
PAH* in Air (μg/m³)	12	0.01	0.0	0.0-0.03	135.3	0.01	
1-OHP in Urine (μmol/mol. Creatinine)	12	0.35	0.21	0.0-1.42	98.8	0.3	
Total:							
PAH* in Air (μg/m³)	38	23.60	0.88	0.0-417.37	165.9	13.4	ND† – 322.9
1-OHP in Urine (μmol/mol. Creatinine)	38	7.46	4.16	0.0-40.90	112.1	2.3	

PAH exposure, assessed via urinary 1-OHP, varied across job tasks. Urinary biomonitoring revealed substantial elevations in 1-OHP, a validated biomarker of internal PAH-dose, among exposed workers. Electrical technicians had the highest mean urinary 1-OHP levels (20.50 μmol/mol creatinine), exceeding the ACGIH BEI of 0.5 μmol/mol creatinine by 41-fold, while mechanical technicians and cleaning workers showed similar elevations (10.59-10.77 μmol/mol creatinine) representing 21-fold exceedances of the BEI, whereas control subjects (0.09 μmol/mol creatinine) and D&R technicians (0.35 μmol/mol creatinine) remained below the regulatory threshold (Figure.1)

Analysis of exposure-biomarker relationships revealed a statistically robust dose-dependent correlation between airborne PAH concentrations and urinary 1-hydroxypyrene (1-OHP) levels among occupationally exposed workers ($r = 0.900$, $p = 1.729 \times 10^{\wedge} (-10)$). This highly significant positive correlation demonstrates that workplace PAH exposure directly influences systemic absorption and subsequent metabolic elimination, confirming the utility of urinary 1-OHP as a reliable biomarker for assessing integrated PAH uptake via the inhalation and dermal routes.

Among the 38 workers evaluated, 26.3% (n=10) exhibited airborne PAH concentrations exceeding the German TRK occupational exposure limit (2 μg/m³), while 63.2% (n=24) presented with urinary 1-OHP concentrations above the American Conference of Governmental Industrial Hygienists (ACGIH) Biological Exposure Index (BEI) threshold (0.5 μmol/mol creatinine). The higher proportion of workers exceeding biological monitoring guidelines than air monitoring thresholds suggests additional exposure pathways beyond inhalation alone, highlighting widespread internal PAH burden across multiple occupational categories and emphasizing the need for comprehensive exposure mitigation strategies.

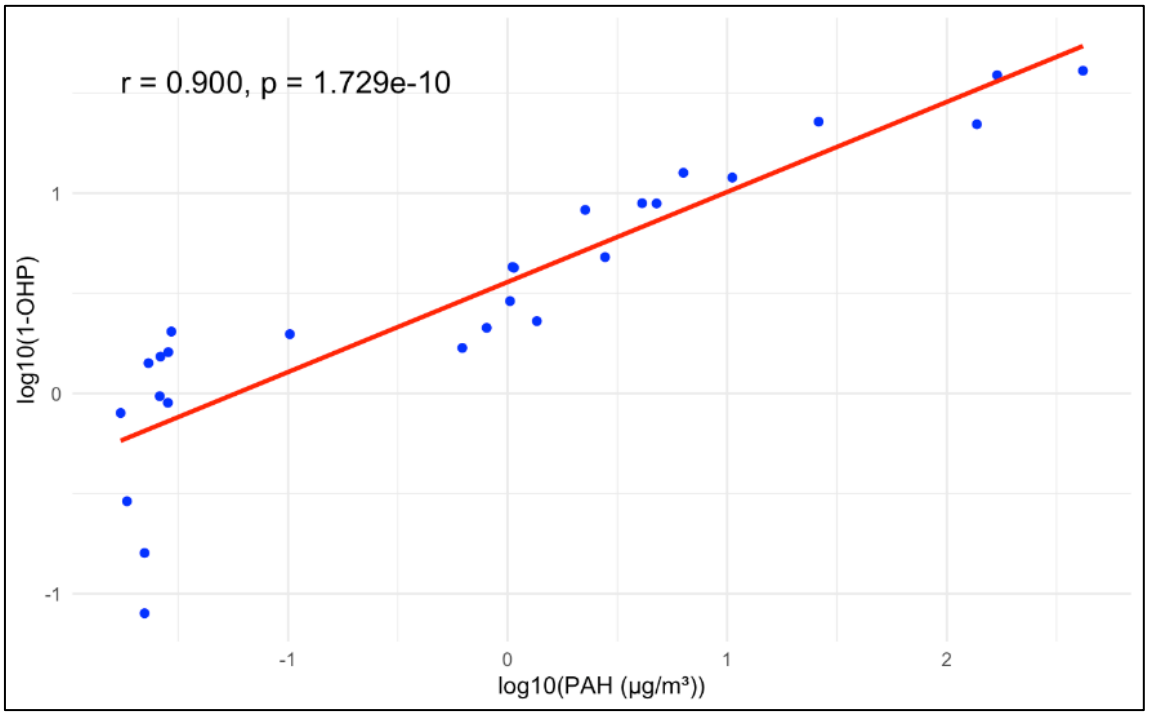

Figure 1: Log–log correlation between airborne PAH exposure concentrations (µg/m³) and urinary 1-hydroxy pyrene (µmol/mol Creatinine) among Aluminum smelter workers

Discussion

Urinary 1-OHP measurements revealed substantial variability in PAH exposure across worker tasks, with electrical workers showing the highest levels due to their direct involvement in PAH-generating processes, such as welding. Mechanical workers had intermittent PAH contact, whereas D&R technicians and controls had minimal exposure due to limited combustion-related activities.[9] Consistent with previous studies showing elevated biomarker levels in welding and industrial maintenance workers,[10] 1-OHP proved to be a reliable indicator of occupational PAH exposure, particularly for airborne compounds.[6] The strong positive correlation between airborne PAHs and urinary 1-OHP (r = 0.900) confirmed a clear dose-dependent relationship, suggesting that workplace PAHs are predominantly low-molecular-weight combustion-derived compounds with inhalation as the primary exposure route. This finding aligns with established research showing rapid metabolism and urinary excretion of inhaled or dermally absorbed PAHs as hydroxylated derivatives,[6] with 1-OHP serving as a reliable biomarker for total PAH exposure, especially lower-molecular-weight compounds from pyrene metabolism, thereby confirming that inhalation exposure is the primary contributor to PAH biomarker levels in these occupational settings.

These findings highlight the necessity for comprehensive workplace exposure controls, encompassing engineering solutions like local exhaust ventilation, consistent use of respiratory protective equipment, and integrated worker hygiene and washing protocols to reduce PAH absorption and associated health risks,[11] while biological monitoring of 1-OHP serves as a practical tool for evaluating cumulative exposure and assessing the effectiveness of implemented control measures.

Limitation

This preliminary investigation sought to evaluate levels of PAH exposure among employees engaged in diverse occupational tasks, thereby providing essential insights for the formulation of effective protective strategies. The robust positive correlation between airborne PAHs and urinary 1-hydroxypyrene (1-OHP) concentrations substantiates the validity of biomonitoring methodologies for assessing occupational PAH exposure in industrial settings. Nonetheless, several limitations must be acknowledged when interpreting the results. The study used urinary 1-OHP as the sole biomarker, which predominantly reflects exposure to low-molecular-weight PAHs and may inadequately reflect exposure to higher-molecular-weight compounds or cumulative bodily exposure. Constraints in sample size, particularly within certain task categories, may have constrained the statistical power necessary to discern subtle exposure variations. Constraints in sample size, particularly within certain task

categories, may have constrained the statistical power necessary to discern subtle exposure variations.

Moreover, potential exposure routes via dermal absorption and ingestion were not quantified, and the spot urine samples employed provided only transient exposure estimates. Additional variables such as dietary patterns, tobacco usage, and environmental PAH exposures external to the workplace were not comprehensively controlled, potentially leading to confounding influences. Given that this study was conducted within a singular aluminum production facility, the results are context-dependent and may not be readily extrapolated to other sectors or geographical locales. Future longitudinal research endeavors that incorporate larger sample sizes, multiple biomarkers, and thorough assessments of exposure pathways are imperative for a more comprehensive understanding of exposure dynamics, associated health risks, and to enhance evidence-based preventive measures. The findings of this investigation will be particularly beneficial for industrial hygienists and occupational health practitioners in devising targeted exposure mitigation strategies and enhancing worker safety in analogous industrial settings.

Conclusion

To conclude, biomonitoring, task-specific exposure assessments, and robust exposure control measures are crucial for safeguarding workers' health and preventing long-term adverse outcomes. With reference to International Occupational Health Guidelines, electrical (mean of 85.25 μg/m^3) and mechanical (50.03 μg/m^3) workers face a consistently high risk of PAH exposure. The mean urinary 1-OHP levels in electrical (average 20.50 μmol/mol Creatinine) and mechanical workers (10.59 μmol/mol Creatinine) exceed the no genotoxic effect benchmark of 1.0 μmol/mol Creatinine by approximately 20 times in electrical and 10 times in mechanical workers, respectively. These levels are far above the background range observed in the general population (0.24-0.76 μmol/mol Creatinine). This highlights the task-specific exposure risks of electrical and mechanical workers, likely due to proximity to combustion sources and inadequate engineering controls. Future efforts will focus on implementing comprehensive industrial hygiene interventions, including optimizing local exhaust ventilation systems, routinely evaluating air filtration efficiency, and strengthening enforcement of PPE compliance. Furthermore, the integration of routine biological monitoring and periodic workplace air sampling will be prioritized to evaluate intervention effectiveness, adapt strategies in real time, and ensure a sustainable reduction in workers' PAH exposure levels.

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