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ORIGINAL ARTICLE

Hydroxylated polycyclic aromatic hydrocarbons as biomarkers of exposure to wood smoke in wildland firefighters

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Wildland firefighter's exposure to wildland fire or vegetative biomass smoke has mostly been assessed by personal monitoring to airborne pollutants. However, the use of biomarkers may accurately reflect the internal (systemic) dose received by the firefighter. In this study, we assessed occupational exposure to wildland fire smoke in 14 wildland firefighters working at prescribed burns at the Savannah River Site, South Carolina by measuring the urinary concentrations of nine hydroxylated metabolites of polycyclic aromatic hydrocarbons (OH-PAHs). Except for 1-hydroxynaphthalene, preshift median concentrations of the OH-PAHs were higher compared with the median concentrations reported among the US general population, indicating elevated exposures to PAHs among the wildland firefighters during the prescribed burn season. The postshift concentrations of OH-PAHs were 83–323% ($P < 0.0001$) higher compared with the preshift concentrations. Higher postshift concentrations of individual OH-PAHs were observed in 49 (87.5%) to 53 (94.6%) of all the 56 pre–post sample pairs. Additionally, the cross-shift (pre- to postshift) increase in 4-hydroxy-phenanthrene urinary concentration was marginally associated ($P < 0.1$) with work shift exposure to $PM_{2.5}$ and significantly associated ($P < 0.05$) with levoglucosan, which is a marker of wildland fire or vegetative biomass smoke. These results suggest that OH-PAHs, especially 4PHE, may be useful biomarkers of wildland fire smoke exposure.

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INTRODUCTION

Smoke resulting from the combustion of vegetation biomass is a significant source of air pollution in indoor and outdoor environments. Exposure to vegetative biomass smoke is common in rural communities in developing countries where most of the estimated 3 billion people who rely on biomass fuels for their residential energy supply live.[1] Wood is one of the more common fuels that is combusted in these communities.[1] However, combustion of wood or vegetation biomass is also an important contributor to air pollution in temperate regions of developed countries where wood is a major source of energy for household heating during the winter.[2–8] Acute wood or vegetative biomass smoke exposure may also occur on a large scale from wildfires.[9,10] Wildland firefighters are occupationally exposed to air pollutants while working to combat wildfires or at prescribed burns. Prescribed burn is often applied as a land management tool to reduce the probability of wildfire occurrence.[11–15]

Vegetative biomass smoke exposure may have significant adverse impact on health.[10,16–18] Vegetative biomass smoke contains many potentially harmful components including particulate matter (PM), carbon monoxide (CO), formaldehyde, acrolein, benzene and polycyclic aromatic hydrocarbons (PAHs).[19] While exposure to wildland fire or vegetative biomass smoke including that of wildland firefighters has been most commonly measured by area measurements or personal monitoring of air concentrations of components (e.g., CO, PM), biological monitoring using a relevant biomarker could also be of interest. The use of appropriate biomarkers is advantageous because they may be more specific to the exposure of concern and therefore may serve as more accurate measures of internal dose. Biological monitoring has been used to assess wildland fire smoke exposure in a few studies. These include the assessment of occupational wildland firefighter wildland fire smoke exposure using exhaled CO, carboxyhemoglobin and urinary methoxyphenols and levoglucosan.[20–23]

Because PAHs have been shown to increase in concentration in indoor and outdoor environments consequent upon the combustion of vegetation biomass,[24–27] it may also be possible to use hydroxylated metabolites of PAHs (OH-PAHs) for biological monitoring of wildland fire smoke exposure among wildland firefighters. PAHs may also be directly connected to the mechanisms through which wildland fire smoke particles induce toxicity through their ability to cause oxidative stress through the formation of oxygenated-PAHs (quinones).[28–30]

OH-PAHs have been used in studies of exposure to and adverse effects of household air pollution resulting from vegetative biomass (including wood) smoke in developing countries.[31–34] In the current study, we used urinary OH-PAHs for biological monitoring of occupational wildland firefighters' wildland fire smoke exposure. We compared the concentrations of OH-PAHs in the wildland firefighters' postshift urine samples and those in samples collected before their work shift at prescribed fires to

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determine whether their work shift wildland fire smoke exposure resulted in exposure to PAHs.

SUBJECTS AND METHODS

Study Location and Subject Recruitment

Savannah River Site (SRS) is a United States Department of Energy National Environmental Research Park located in South Carolina. The park's forest resource comprising of 31% hardwood or mixed pine/hardwood and 69% pine is managed by the United States Forest Service. A resident crew of United States Forest Service wildland firefighters annually applies prescribed burns to ~20,000 acres of forest within SRS to reduce forest fuel load and restore ecological processes.[35] A total of 19 (17 males and 2 females) wildland firefighters who worked at prescribed burns at SRS participated in an exposure assessment study during the dormant winter seasons (January–March) of 2008 and 2009.[13] No respiratory protection was worn by the subjects while they conducted prescribed burns as is currently typical of wildland firefighters.[11] The subjects were recruited into the study after the objectives of the study were explained to them, and they had signed the study consent form. The study was approved by the University of Georgia Institutional Review Board for the inclusion of human subjects. The involvement of the Centers for Disease Control and Prevention (CDC) laboratory was determined not to constitute engagement in human subject research.

Personal exposures of the subjects to CO and $PM_{2.5}$ (PM with median aerodynamic diameter of 2.5 μm) were monitored across 30 prescribed burn work shifts. Spot preshift urine samples were collected for all sampled subject-work shifts immediately before work at prescribed burns. Spot postshift samples were collected on the same day immediately after prescribed burn was completed. For the current study, 56 pre- and postshift urine sample pairs from 14 subjects (12 males and 2 females) working at 16 of the prescribed burn work shifts with an average duration of 7.6 h were identified for OH-PAH biomonitoring. The urine sample pairs were selected based on the following criteria: (1) they were collected from the wildland firefighters at prescribed burns occurring at least two days since they had last worked at a prescribed burn or wildfire, and (2) they had corresponding personal $PM_{2.5}$ exposure data. Additionally, all but four of the urine sample pairs had corresponding PM-associated levoglucosan data, and all but three had corresponding CO exposure data. The urine samples were coded after collection, immediately frozen at − 20 °C on site and later shipped on dry ice to the CDC in Atlanta and were stored at − 70 °C until analysis.

Personal $PM_{2.5}$, Levoglucosan and CO Monitoring

The protocols for the personal monitoring of exposure to $PM_{2.5}$, particle-associated levoglucosan and CO along with the associated results have been reported elsewhere.[13] Briefly, work shift $PM_{2.5}$ exposure was gravimetrically determined at the University of Georgia. Particles were collected on Gelman 37 mm polytetrafluoroethylene filters loaded into a BGI triplex cyclone (BGI, Waltham, MA, USA) that were connected to SKC Air Check Model 2000 pumps (SKC, Eighty Four, PA, USA). The pumps were set to flow at 1.5 l/min. Personal work shift CO exposure was monitored in real time using Drager Pac III data-logging single gas monitors (Drager Safety, Pittsburgh, PA, USA).

Particle-associated levoglucosan was analyzed at the University of Washington by gas chromatography/mass spectrometry as described previously.[13] Briefly, the PM sample filters were spiked with the recovery standard, deuterated levoglucosan and extracted by sonication in a freshly prepared solution of 3.6 mM triethylamine in ethyl acetate. The extracts were reduced to ~0.5 ml under nitrogen and filtered through a 0.45 μm polytetrafluoroethylene syringe into silanized autosampler vials. After the addition of the internal standard (triisopropylbenzene) and anhydroheptulose, used to monitor the efficiency of derivatization, the extracts were derivatized by the addition of methylsilyltrifluoroacetamide together with trimethylchlorosilane and pyridine and allowed to react in the dark for 6 h. The extracts were then injected into the GC by splitless injection (GC Column: RESTEK Rtx-5Sil MS; RESTEK Corp., Bellfonte, PA, USA). Quantification was carried out using the *m/z* peaks of 204 (levoglucosan) and 206 (deuterated levoglucosan) relative to the response at *m/z* 180 (triisopropylbenzene).

PAH Biomonitoring

In the urine, we measured 9 mono-hydroxylated PAHs: 1-, 2-hydroxynaphthalene (1NAP, 2NAP), 2-, 3-hydroxyfluorene (2FLU, 3FLU), 1-, 2-, 3-, 4-hydroxyphenanthrene (1PHE, 2PHE, 3PHE, 4PHE) and 1-hydroxypyrene (1PYR). Analyses were carried out using online solid-phase extraction coupled with high-performance liquid chromatography tandem mass spectrometry.[36] In summary, 0.2 ml urine sample was spiked with a mixture of ^{13}C-labeled internal standards before sample preparation, and 0.1 ml of 1 M sodium acetate buffer (pH = 5.5) containing β-glucuronidase/arylsulfatase enzyme from *Helix pomatia* and ascorbic acid solution. The mixture was then allowed to incubate at 37 °C overnight, which deconjugated the glucuronide and sulfate conjugates of the PAH metabolites present in urine and yielded free OH-PAHs. The samples were spiked with 175 μl of methanol and centrifuged. The supernatant of the sample mixture was diluted with 350 μl of deionized water before instrumental analysis. Analytical determination of the target analytes were performed by online solid-phase extraction coupled with high-performance liquid chromatography tandem mass spectrometry. The instrumentation setup includes an iChrom Symbiosis online solid-phase extraction system, an Agilent 1260 high-performance liquid chromatography and an AB Sciex 5500 mass spectrometer operated under negative electrospray ionization mode. The limits of detection for the analytes are as follows: 80 ng/l for 1NAP and 2NAP, 50 ng/l for 3FLU and 1PYR and 20 ng/l for the remaining OH-PAHs.

Urinary creatinine was measured at CDC on a Roche Hitachi 912 Chemistry Analyzer (Hitachi, Pleasanton, CA, USA) using the Creatinine Plus Assay (Roche Diagnostics, Indianapolis, IN, USA).

Questionnaire and Time-Activity Diary

Information regarding the size of the area burned (acres) during the subject's prescribed burn work shift, length of work shift and tobacco smoking, secondhand smoke (SHS) exposure, and grilled and smoked food consumption within the prior 48 h was obtained using a self-administered questionnaire filled out by the subjects after each work shift. A time-activity diary was also filled out after each prescribed burn work shift and was used to record the task that the subjects performed while at the fire line. Tasks were classified according to the three main tasks performed at the fire line (lighting, holding or mop-up) if the subject had spent at least 75% of the time at the fire line performing the task. Lighting involves the ignition of the vegetative biomass fuel; holding involves activities geared towards maintaining the fire within the boundaries of the area being burned; mop-up activities entail the extinguishing of smoldering fire after the fuel has been consumed. Tasks were categorized as "unclassed" when the wildland firefighters spent most of the time on other tasks or did not spend up to 75% of their time at the fire line performing any one task.

Statistical Analysis

Concentrations of OH-PAHs below the limits of detection were replaced with the limits of detection divided by the square root of 2.[37] Creatinine correction (the concentration of OH-PAH in urine divided by the concentration of creatinine in urine) was applied to OH-PAH concentrations before they were used in statistical analyses to account for urine dilution. Linear mixed-effect models were first used to test whether the postshift creatinine-corrected concentrations of the individual OH-PAHs and of their sum (Σ(OH-PAHs)) were increased compared with the preshift concentrations. The response variables in the models were the log-transformed urinary concentrations of the OH-PAHs, whereas the predictor variable was the time of urine collection (pre- or postshift). The possible effects of various factors on the cross-shift differences (postshift minus preshift concentrations) in the log-transformed OH-PAH urinary concentrations were then individually tested using linear mixed-effect models. These factors include the occurrence of SHS exposure during the work shift, the task that the subject performed most of the time at the fire line (> 75%), consumption of grilled or smoked food within 48 h before sampling, the size of burn, the length of the work shift, age and work shift exposures to CO, $PM_{2.5}$ and levoglucosan. The random subject effects were included in the models to control for correlations between multiple measurements collected from the same subject, and random effects for the sample collection date were included to control for correlation between urinary OH-PAH concentrations in samples collected on the same day.

All statistical analyses were performed using the SAS version 9.3 (SAS Institute, Cary, NC, USA). The linear mixed-effect models were run using the PROC MIXED procedure in the software.

Table 1. Pre- and postshift creatinine-corrected OH-PAH concentrations (ng/g creatinine) and their ratios in firefighter urine samples ($n = 14$).[a]

OH-PAH	*Estimate (95% confidence limits)*			*Proportion of all subject-days with higher postshift concentrations (% out of 56)*
	Preshift concentration	*Postshift concentration*	*Post/pre ratio*	
1NAP[b]	2085 (1305, 3331)	8824 (5523, 14,098)	4.23 (2.93, 6.11)	94.6
2NAP[b]	4046 (3071, 5331)	12,072 (9163, 15,905)	2.98 (2.43, 3.67)	91.1
2FLU[b]	496 (371, 663)	1491 (1115, 1994)	3.01 (2.48, 3.65)	89.3
3FLU[b]	199 (150, 264)	426 (321, 564)	2.13 (1.74, 2.62)	92.9
1PHE[b]	247 (183, 334)	557 (412, 752)	2.25 (1.89, 2.25)	92.9
2PHE[b]	123 ((94, 163)	346 (262, 457)	2.81 (2.33, 3.37)	94.6
3PHE[b]	198 (150, 263)	705 (531, 935)	3.55 (2.86, 4.41)	91.1
4PHE[b]	33 (25, 43)	121 (92, 158)	3.65 (2.88, 4.61)	92.9
1PYR[b]	313 (212, 463)	576 (390, 851)	1.83 (2.18, 1.55)	87.5
∑(OH-PAHs)[b]	9202 (6828, 12,402)	27,447 (20,406, 37,064)	2.99 (2.39, 3.74)	91.1

Abbreviations: 2FLU, 2-hydroxyfluorene; 3FLU, 3-hydroxyfluorene; OH-PAH, hydroxylated polycyclic aromatic hydrocarbons; 1NAP, 1-hydroxynaphthalene; 2NAP, 2-hydroxynaphthalene; 1PHE, 1-hydroxyphenanthrene; 2PHE, 2-hydroxyphenanthrene; 3PHE, 3-hydroxyphenanthrene; 4PHE, 4-hydroxyphenanthrene; 1PYR, 1-hydroxypyrene. [a]Comparisons of pre- and postshift concentrations were carried out using linear mixed-effects model adjusting for subject and sample collection date as random effects. [b]Geometric mean postshift concentration of the urinary OH-PAH was significantly higher ($P < 0.0001$) than the geometric mean preshift concentration.

RESULTS

Geometric mean concentrations of the exposures of the wildland firefighters to CO, $PM_{2.5}$ and levoglucosan at the prescribed burns when the urine samples used for this study were collected were 1.34 p.p.m. (confidence limits: 1.07, 1.67 p.p.m.), 577 $\mu g/m^3$ (492, 675 $\mu g/m^3$) and 21 $\mu g/m^3$ (14, 30 $\mu g/m^3$), respectively. The size of the area treated with prescribed burn varied between 170 to 2505 acres, with an average of 847 acres, whereas the length of the work shift of the subjects was between 5.5 and 9.5 h, with an average of 7.6 h.

All the OH-PAHs were above the limits of detection in $>96\%$ of the samples, with 2NAP, 2FLU, 1PHE and 1PYR being detected in all the samples. The model-derived geometric means of the creatinine-corrected OH-PAH concentrations are presented in Table 1. 2NAP was the OH-PAH with the highest concentrations, whereas 4PHE had the lowest concentrations in both pre- and postshift urine samples among the subjects. Postshift urinary concentrations of all individual OH-PAHs and the ∑(OH-PAHs) were significantly higher compared with the preshift concentrations ($P < 0.0001$; Table 1), with the postshift concentrations being 1.83–4.23 times higher compared with the preshift concentrations. The creatinine-corrected postshift concentration of ∑(OH-PAHs) was 2.99 times higher than the preshift concentration.

Work shift exposure to CO and $PM_{2.5}$, size of the prescribed burn, length of work shift and the task performed by the subject during the prescribed burn had no effect on the cross-shift (postshift minus preshift) differences of the individual creatinine-corrected OH-PAHs concentrations or the corresponding difference in the creatinine-corrected ∑(OH-PAH) concentrations in linear mixed-effect models (with adjustment for only date of sample collection and subject as random variables and adjustment for no other factor). Two of the subjects who had only a total of 3 subject-days of samples reported being current smokers. However, only one of the subjects smoked (once) during the work shift when the urine samples were collected. Therefore, the effect of smoking on the cross-shift differences in OH-PAH concentrations was not tested. Subjects reported having SHS exposures on 16 subject-days. Subjects also reported eating smoked or grilled foods within the previous 48 h on eight occasions. Cross-shift differences were higher for all OH-PAHs when the subjects were exposed to SHS during the work shift, and lower when they had eaten smoked or grilled foods within the previous 48 h. However, these differences were not statistically significant. The relationship between age and cross-shift differences in the OH-PAHs was inconsistent and largely insignificant. A significant association with age was only observed for 1PYR with increasing cross-shift difference (0.029 log units) with age ($P = 0.0424$).

Only cross-shift difference in creatinine-corrected 4PHE concentration was positively associated with work shift exposure to levoglucosan ($P = 0.0062$) in linear mixed-effect models adjusted for the date of sample collection and subject as random variables. The relationship remained significant when the model was adjusted for SHS exposure, task of the subject during the prescribed burn, size of the burn, length of the work shift and eating of smoked or grilled food within the previous 48 h ($P = 0.0262$). The relationship between cross-shift differences in creatinine-corrected 4PHE concentration and work shift exposure to $PM_{2.5}$ adjusted for only date of sample collection and subject as random variables was marginally significant ($P < 0.10$) in linear mixed-effect model. Marginal significance was also observed for the relationship between cross-shift differences in creatinine-corrected 1NAP concentration and work shift exposure to levoglucosan in a similar model. However, the marginal significance of these two relationships was not sustained when other factors were adjusted for in the models.

DISCUSSION

The average CO concentration in the personal air sample of the firefighters who provided the urine samples used for the OH-PAH analyses (14 subjects; 56 person-days) is comparable to the average concentration observed across the entire study (18 subjects; 157 person-days) from which the samples were selected: 1.3 *versus* 1.5 p.p.m., respectively. Average personal air sample concentrations of $PM_{2.5}$ (577 *versus* 530 $\mu g/m^3$) and levoglucosan (21 *versus* 20 $\mu g/m^3$) were also comparable between the two groups.[13] The personal air sample concentrations of CO and $PM_{2.5}$ were also within the range of levels observed during a previous wildland firefighter exposure study at SRS,[12] and comparable to time-weighted averages recorded in studies in the Western United States.[11]

The substantial and significant higher postshift creatinine-corrected urine concentrations of the nine OH-PAHs (1.83- to 4.23-fold increase) compared with the preshift concentrations in the current study suggest that the occupational exposure of the wildland firefighters at prescribed burns to elevated levels of wildland fire smoke, as demonstrated by their work shift personal exposures to $PM_{2.5}$, contributed to their PAH exposures. Concentrations of most of the OH-PAHs were at least doubled across the work shift, except 1PYR (1.83-fold increase). This could be expected because emissions of PAHs from wood combustion are

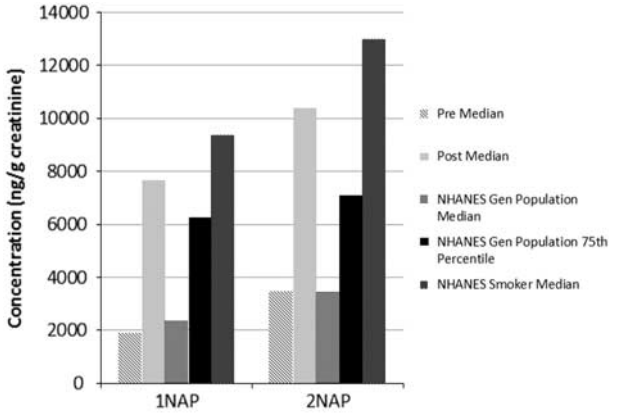

Figure 1. Comparison of geometric mean pre- and postshift creatinine-corrected hydroxyl-naphthalene metabolite concentrations with reported geometric mean and 75th percentile concentrations in the US adult (⩾20 years) population (2007–2008 NHANES) and adult (20–49 years) smokers (2011–2012).

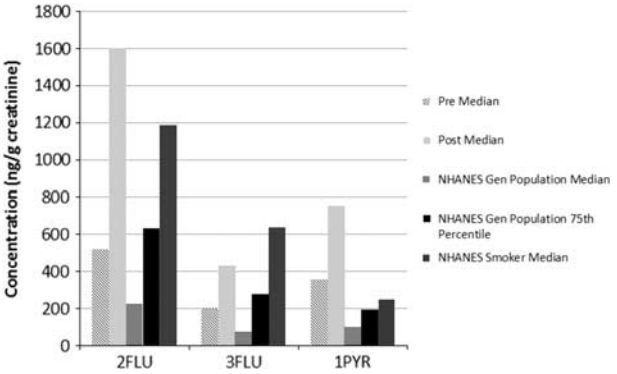

Figure 2. Comparison of geometric mean pre- and postshift creatinine-corrected hydroxyl-fluorene and pyrene metabolite concentrations with reported geometric mean and 75th percentile concentrations in the US adult (⩾20 years) population (2007–2008 NHANES) and adult (20–49 years) smokers (2011–2012).

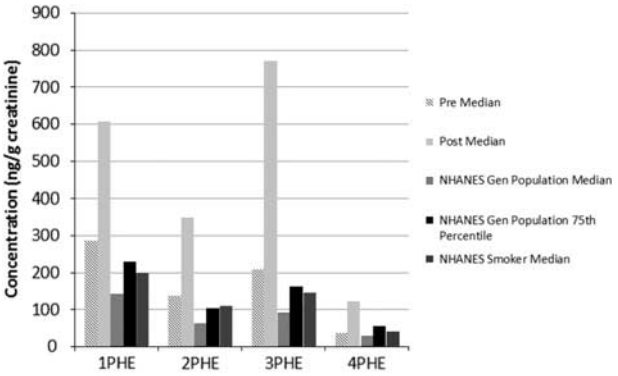

Figure 3. Comparison of geometric mean pre- and postshift creatinine-corrected hydroxyl-phenanthrene metabolite concentrations with reported geometric mean and 75th percentile concentrations in the US adult (⩾20 years) population (2007–2008 NHANES for 1PHE, 2PHE, 3PHE and 2005–2006 NHANES for 4PHE) and adult (20–49 years) smokers (2011–2012).

relatively high compared with those from less clean fuel such as coal briquette and kerosene.[38,39] Wood/vegetative biomass combustion has also been identified as a major source of atmospheric PAHs,[40,41] and is a dominant contributor to the ambient air concentrations of PM and PAHs in certain areas in developed countries where wood is a major heating fuel.[2–8,25,26] Furthermore, it has been shown in studies that PAH content is higher in wood smoke particles compared with particles from other common sources of ambient air pollution such as traffic and diesel exhaust.[42,43]

Although 1PYR had been commonly used as a biomarker of PAH exposure, it had the least percentage increase (83%) among the nine OH-PAHs that were examined in the current study. The largest percentage increase (323%) from pre- to postshift was observed for 1NAP. Pre- to postshift increases above 200% were also observed for 4PHE (265%), 3PHE (255%) and 2FLU (201%). Similarly, 2NAP had been observed to be more sensitive to exposure to vegetative biomass smoke compared with 1PYR among charcoal production workers in Brazil.[44] There was a 198% increase in 2NAP in the current study. The significant association that was observed between cross-shift changes in 4PHE and work shift levoglucosan exposure in both the simple and more complex models also supports the observation that other OH-PAHs may be more appropriate as biomarkers of wildland fire or vegetative biomass smoke exposure compared with 1PYR. Although the effects of SHS exposure during the work shift (increased cross-shift changes) and consumption of smoked and/or grilled food within the prior 48 h (decreased cross-shift changes) were in the expected direction,[45] their associations with the changes in OH-PAHs were not significant. Additionally, controlling for them and other factors did not change the association between 4PHE and levoglucosan exposure.

Consequently, biomonitoring as assessed in the current study could serve as an alternative approach for evaluating occupational wildland fire smoke exposure of the wildland firefighter instead of personal air monitoring as was mostly carried out in previous studies.[11–15] Unlike in the current study, previous use of urinary OH-PAHs for biomonitoring vegetative biomass smoke exposure has mainly been within the context of chronic exposure to elevated levels of wood smoke in indoor environments and/or interventions to reduce such exposures.[31,33,34] Urinary concentrations of 2NAP and 1PYR have also been used to assess wood smoke exposure among workers in a charcoal production plant.[44]

Although substantial pre- to postshift increases in the OH-PAHs levels among the subjects indicated occupational PAH exposure, it should be noted that wildland firefighters are potentially exposed to other sources of PAHs apart from wildland fire smoke at prescribed burns. However, the median preshift creatinine-corrected concentrations of OH-PAHs, except 1NAP, appear higher compared with the median concentrations (Figures 1–3) observed for the United States adult population (20 years) in the 2007–2008 (2005–2006 for 4PHE) National Health and Nutrition Examination Survey (NHANES).[46] Additionally, the median preshift creatinine-corrected concentrations of 1PYR, 1PHE, 2PHE and 3PHE were all apparently higher compared with the 75th percentile concentrations for the United States adult population and median concentrations among adult (20–49 years) smokers in the United States.[46] The median postshift OH-PAH concentrations exceeded the 95th percentile of concentrations of hydroxylated phenanthrene (except 4PHE; >90th percentile) and pyrene metabolites observed in the general population in the survey.[46] These results indicate that the subjects' PAH exposures during the prescribed burn seasons when the samples were collected were above the average exposures of the general population in the country.

Sources of PAH exposures apart from wildland fire smoke for the wildland firefighter at prescribed burns include vehicle exhaust and the lighting fuel (1 part gasoline to 3 parts of diesel mixture) used to ignite the biomass. However, it is not expected that these would have affected the overall results regarding the

higher postshift creatinine-corrected concentrations of OH-PAHs. Emissions from these sources are small compared with those from the combustion of biomass at prescribed burns, and cross-shift changes in the OH-PAHs were not different between the firefighters who were lighting with the gasoline/diesel lighting fuel and those performing other tasks. Cigarette smoke is also a source of PAHs.[45] However, cigarette was smoked by one subject during the work shift only once throughout the study period, and exposure to SHS during the work shift was associated with an insignificant higher cross-shift increases in the urinary OH-PAHs. It should be noted that the insignificance of this result could have been due to the small sample size of the study.

Other possible sources of exposure away from work including the use of wood for heating at homes and dietary sources could also have affected the results.[24,45] However, only one of the subjects reported using wood as an energy source for residential heating and so this variable was not controlled for during statistical analyses. Consumption of grilled or smoked food within the previous 48 h did not have any effect on cross-shift differences in urinary OH-PAH concentrations. Moreover, unreported exposure to such sources would have likely biased the results towards the null as they would have likely contributed towards an increase in the preshift measurements and a reduction in cross-shift increases in the OH-PAHs. Furthermore, the significant pre- to postshift increases in OH-PAHs were consistent with higher postshift creatinine-corrected concentrations of the analytes being observed in 49 (87.5%) to 53 (94.6%) of the 56 subject-days of measurements (Table 1). The collection of spot urine immediately after the work shift at prescribed burns can also be viewed as a limitation of the study since peak excretion of OH-PAHs in urine occurs a few hours after exposure. The average time of maximum excretion of the OH-PAHs that were measured in this study occurred 4.6 to 8.3 h after experimental exposure to wood smoke.[47] Nonetheless, timing spot urine collection to match peak excretion would have rather strengthened the results observed in this study.

In conclusion, there were consistent substantial increases in OH-PAHs across prescribed burn work shifts among the wildland firefighters in this study. Additionally, cross-shift changes in creatinine-corrected 4PHE was associated with personal exposure to airborne pollution (significantly with levoglucosan and marginally with $PM_{2.5}$). The results suggest that OH-PAHs, especially 4PHE, may be applicable as biomarkers of wildland fire or vegetative biomass smoke exposure in elevated exposure situations in environments with relatively low background PAH levels.

CONFLICT OF INTEREST

The authors declare no conflict of interest.

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DISCLAIMER

The findings and conclusions in this report are those of the authors and do not necessarily represent the views of the Centers for Disease Control and Prevention (CDC). The use of trade names and commercial sources is for identification only and does not constitute endorsement by the US Department of Health and Human Services or CDC.

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