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Wildfire Impact on Indoor and Outdoor PAH Air Quality

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ABSTRACT: Air quality impacts from wildfires are poorly understood, particularly indoors. As frequencies increase, it is important to optimize methodologies to understand and reduce chemical exposures from wildfires. Public health recommendations use air quality estimates from outdoor stationary air monitors, discounting indoor air conditions, and do not consider chemicals in the vapor phase, known to elicit adverse effects. We investigated vapor-phase polycyclic aromatic hydrocarbons (PAHs) in indoor and outdoor air before, during, and after wildfires using a community-engaged research approach. Paired passive air samplers were deployed at 15 locations across four states. Twelve unique PAHs were detected only in outdoor air during wildfires, highlighting a PAH exposure mixture for future study. Heavy-molecular-weight (HMW) outdoor PAH concentrations and average Air Quality Index (AQI) values were positively correlated ($p < 0.001$). Indoor PAH concentrations were higher in 77% of samples across all sampling events. Even during wildfires, 58% of sampled locations still had higher indoor PAH air concentrations. When AQI values exceeded 140 (unhealthy for sensitive groups), outdoor PAH concentrations became similar to or higher than indoors. Cancer and noncancer inhalation risk estimates from vapor-phase PAHs were higher indoors than outdoors, regardless of the wildfire impact. Consideration of indoor air quality and vapor-phase PAHs could inform public health recommendations regarding wildfires.

KEYWORDS: *passive sampling, vapor-phase PAHs, public health, community-engaged research, wildfire PAH exposure mixture*

INTRODUCTION

Wildfire frequencies are increasing in some regions under a warming climate.[1] In 2017, the Western United States (U.S.) had the second worst wildfire season on record, resulting in over 10 million acres burned.[2] 2020 was the most active wildfire year on record for the Western U.S. Over six million acres burned in California, Oregon, and Washington. Six of the 20 largest California wildfires occurred,[3] and nearly 4,000 more homes were lost in Oregon than in the previous 5 years combined.[4,5] Many cities across these three states saw historically poor air quality across multiple days.[6,7]

While the number of wildfires each year has been fairly consistent, the number of large wildfires (>1,000 acres) has increased by seven fires per year[8] and the total acreage burned and the average size has increased.[9,10] Since 1970, the wildfire season in the Western U.S. is 105 days longer, and high wildfire potential days are predicted to increase by 6−34 days by 2050.[11] As impacts increase, it is important to understand and reduce chemical exposures from wildfires.

Wildfire smoke is a complex mixture with knowledge gaps regarding its composition and health impact.[12] One class of concern is polycyclic aromatic hydrocarbons (PAHs), a widespread organic contaminant arising from both natural and anthropogenic sources with links to cancers, developmental and neurological issues, respiratory problems, and suppressed immune functions.[13−15] In general, low-molecular-weight (LMW) PAHs (two or three rings) tend to be more acutely toxic, while high-molecular-weight (HMW) PAHs (4-ring and above) are usually more carcinogenic.[16,17]

Current wildfire public health messaging is based on risks from particulate matter and U.S.-regulated chemicals.[18] However, PAHs are semivolatile chemicals and are present in both the vapor phase and bound to some types of particulate matter.[19] Previous studies have shown that vapor-phase PAHs can account for up to 86% of the cancer risk from total inhalation exposure (vapor phase and particulate matter).[20−23] Understanding the distributions of vapor-phase PAHs is important for more comprehensive public health recommendations.

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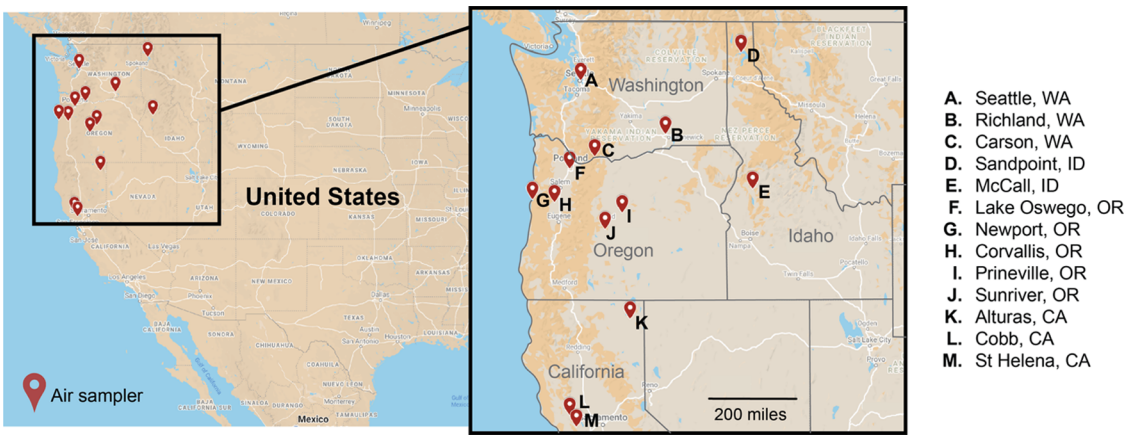

Figure 1. Sampling locations across four Western U.S. states.

Wildfire public health messaging also does not account for indoor air. In a study by Messier et al.,[24] passive samplers were deployed indoors and outdoors at six urban sites in Eugene, Oregon, across 7 days during the 2018 wildfire season. Results showed that indoor PAH air concentrations were consistently equal to or greater than outdoor concentrations, even during periods of mild smoke impact (AQI 0−100). Messier et al.[24] also found that only outdoor PAH air concentrations were associated with satellite-based wildfire smoke density. This study builds on the approach by Messier et al.[24] to better understand the fate of, and potential human exposure to, vapor-phase PAHs from wildfires in indoor and outdoor air. Study objectives were to (1) compare PAH air concentrations before, during, and after wildfire sampling events; (2) compare indoor versus outdoor air concentrations; (3) assess the influence of wildfire characteristics (such as wildfire size and smoke density) and participant behaviors (woodstove use, ventilation) on indoor and outdoor PAH concentrations; and (4) explore the influence of wildfires on cancer and noncancer inhalation risks.

MATERIALS AND METHODS

Sampling Locations. This study was performed from August to November in 2018, 2019, and 2020. Over these 3 years, passive sampling took place at 15 locations in the Western U.S. across Washington, Oregon, California, and Idaho (Figure 1). Passive sampling devices (PSDs) non-selectively sequester organic compounds in a biomimetic manner.[25] Their ease of implementation and low maintenance make passive samplers an ideal approach for community-engaged research (CEnR). Through CEnR, researchers can work with trained community members across a large geographical area to rapidly deploy samplers.

Participants (18 or older) were selected using a convenience sampling design based on residence in high-risk areas for wildfires in the Western U.S. Prospective participants were contacted by the study coordinator and informed of the purpose of the study, duration, and the activities for which they would be responsible.

Participants received a kit containing the PSDs, an instruction packet, a survey, and a prepaid return label. At all locations, one passive air sampler was placed inside the home and a second sampler was placed outside the property. At one site, samplers were deployed in triplicate to assess field variability, and a field trip blank was included to assess potential contamination during the transportation process. PSDs were deployed for 3−4 weeks and then mailed back to the Food Safety and Environmental Stewardship Lab at Oregon State University.

Given the duration of the study, participant retention was important. An initial 13 locations were identified in 2018. Over time, some participants had a lack of interest (n = 3), were unavailable during some data collections (n = 2), or moved out of the state (n = 2). Where possible, new participants were recruited to fill spots, resulting in 15 unique locations across 13 cities. Overall, participant retention across the 3 years of the study was 62%. Locations were only included in the final data set if there were at least two different sampling events captured. Twelve of the 15 locations were sampled during wildfires.

Sample Preparation. One meter passive sampling strips were constructed from additive-free low-density polyethylene (LDPE) (Burntwood Plastics Ltd.) and cleaned with hexanes, as previously described.[26,27] Three performance reference compounds, fluorene-d_{10}, pyrene-d_{10}, and benzo[b]-fluoranthene-d_{12}, were added to the LDPE prior to deployment to allow for calculations of *in situ* uptake rates. The average of three blank-infused LDPE strips was used to determine t = 0 performance reference compound concentrations (Table S1). The analyte sampling rate was derived from performance reference compound loss.

At all locations, T-shaped air cages[28,29] containing five LDPE strips were placed in a room inside and outside the home. Indoor samplers were placed between the floor and the adult breathing-zone height (1−2 meters). Outdoor air samplers were placed approximately 1 meter above the soil. Temperature loggers were placed inside all air cages for the duration of the deployment period.

LDPE samples were transported in sealed polytetrafluoroethylene bags at ambient temperatures, and once at the laboratory, they were stored at −20 °C.[30−32] Following established methods, samples were cleaned using isopropanol to remove particulate matter and moisture, extracted in *n*-hexane, quantitatively transferred, and then concentrated, as previously described.[27−29,33] Prior to extraction, surrogates (naphthalene-d_8, acenaphthylene-d_8, phenanthrene-d_{10}, fluoranthene-d_{10}, chrysene-d_{12}, benzo[a]pyrene-d_{12}, and benzo[ghi]perylene-d_{12}) were spiked onto samples for the calculation of surrogate recovery. Sample extracts were stored at −20 °C. Solvents were Optima grade (Fisher Scientific,

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Pittsburg, PA) or equivalent. Deuterated surrogates were from CDN Isotopes (Pointe-Claire, Quebec Canada).

QC Samples. A standard containing all target analytes was used to verify the continuing calibration during analysis. Laboratory quality control (QC) samples including construction, lab processing, cleaning, reagent, and instrument blanks as well as sample duplicates and overspikes were used to ensure data quality (Table S2).

GC-MS/MS. Analysis for 65 PAHs was performed with an Agilent, a 7890A gas chromatograph (GC), a 7000C triple quadrupole mass spectrometer (MS/MS), and a select PAH column (30 m × 250 μm × 0.15 μm).[34] At least a 3-point (3−7 points) calibration was employed with correlations ≥0.99.[34] The PAH physicochemical properties and instrument parameters are detailed in Tables S3 and S4. GC-MS/MS data was analyzed using the MassHunter Quantitative Analysis v.B.06.00 SP1 build 6.0.388.1 (Agilent Corp. Wilmington, DE) software. The Agilent GC-MS/MS method conducts automatic surrogate correction to account for any losses during sample processing in the laboratory. The average extraction surrogate recovery was 87% (59−109%). Perylene-d_{12} was used as the instrumental internal standard. Nine of the 65 PAHs were not detected in any samples and were excluded from further analysis.

Calculations. Time-weighted average concentrations for the air vapor phase were determined using an empirical uptake model and *in situ* sampling rates derived from performance reference compounds, as described by Huckins et al.[25,35] The sampler−air partitioning coefficient is temperature-corrected using the average temperature during deployment. Detailed equations are provided in SI Page S5.

The quantitative human health risk assessment was used to estimate the excess lifetime cancer risk and noncancer hazard from inhalation exposure to vapor-phase PAH mixtures in indoor and outdoor air across all sampling events[36−38] (SI Page S5 and Table S5). Risk assessment values calculated in this study account for risk to vapor-phase PAHs only. Additionally, reference values to estimate the inhalation cancer risk were available for only 20 of the 65 PAHs in our analytical method.[38−43] Just four reference concentration (RfC) values, used for noncancer hazard calculations, are published for PAHs.[38,42] Values for other PAHs are not available due to lack of or insufficient data. Additionally, most of the published RfC values are for 2-ring PAHs (naphthalene, 1-methylnaphthalene, 2 methylnaphthalene, and benzo[*a*]pyrene) and do not reflect risk from higher-molecular-weight PAHs. Therefore, the risk assessments provided in this study merely represent a fraction of the entire PAH mixture.

Wildfire Characteristics and Participant Behaviors. At each location, the U.S. Environmental Protection Agency (EPA) Air Quality Index (AQI) values for particulate matter (based on $PM_{2.5}$ and PM_{10} combined) were obtained for each day of sampling using the closest available EPA monitor.[44] The average distance of participant locations to AQI monitors was 15 miles (2−54 miles). AQI values are sectioned into six different categories: 0−50 "good", 51−100 "moderate", 101−150 "unhealthy for sensitive groups", 151−200 "unhealthy", 201−300 "very unhealthy", and 301−500 "hazardous".[45] Wildfire smoke density was obtained for each day of sampling using the National Oceanic and Atmospheric Administration satellite-based model for wildfire smoke called the Hazard Mapping System (HMS).[46] During wildfire impact, information for all wildfires near sampling locations was collected. AQI values, distance from AQI monitor, smoke density, and wildfire information are provided in Tables S6−S12.

Following each sampling event, participants were asked to complete a survey. The survey format used in 2018 consisted of basic questions related to the location of the air samplers, the opening of windows, the use of air conditioning, and participants' interest in receiving results from their residence. The basic survey was expanded in 2019 and 2020 and transitioned online (Qualtrics XM Survey Software). Specifically, questions were included to identify potential PAH inhalation exposure sources other than wildfires, such as any use of or exposure to the following: air fresheners, candles/incense, use of a fryer, broiler or charcoal grill, wood-fired heating sources, smoker status (cigarette, e-cigarette, cigar), and type of kitchen stove (gas, electric). The 2019 and 2020 survey activities were conducted under the Institutional Review Board approval from Oregon State University (protocol # IRB-2019−0312). Survey compliance for basic survey questions was 79% across all 3 years. Survey compliance for expanded survey questions in 2019 and 2020 was 55%. Survey results are provided in Table S13.

Following each round of sampling, individual data sets were returned to study participants. The reports included a comparison of different PAH concentrations indoors and outdoors for each location, overall study conclusions, common sources of PAHs, and ways to reduce exposure. The 2020 reports were further expanded to include results from each sampling year to demonstrate trends in air quality across years and to evaluate air quality based on wildfire status.

Statistical Analysis and Regression Modeling. Samples were binned into one of three categories: before wildfire (10−12 months since any wildfire activity), during wildfire, and after wildfire (about 1 month after wildfire) based on wildfire activity during deployment. All statistical analyses were performed using R 3.5.2 and JMP Pro 14.0.1 statistical software. Statistical analyses were only completed for compounds that were above detection limits during at least one sampling event. Analytes below method quantification limits were assigned 1/2 method detection limits (Tables S14−S16). The relative standard deviation was calculated based on n = 3 replicates[24,26,47−50] at Newport, Oregon (Table S17).

The mean PAH concentrations in air before wildfire versus wildfire, and wildfire versus after wildfire, were compared at each location using a one-sided t-test. Data was analyzed by grouping PAHs by ring size to assess trends based on the PAH physicochemical and potential toxicological properties. Two- and 3-ring PAHs are defined as low molecular weight (LMW), while 4-ring and above are defined as high molecular weight (HMW). Statistical analysis of 5-ring and 6-ring PAH air concentrations during wildfire compared to that of no wildfire or after wildfire was not completed for seven locations due to nondetect frequency or only n = 1 PAH was detected. The mean PAH concentrations in indoor and outdoor air at each sampling event were compared using a one-sided t-test. For all statistical analyses, concentrations were log-transformed, and a Bonferroni procedure was used to adjust the significance level and reduce the probability of type I errors.

A simple linear regression model was used to explore the influence of AQI values, NOAA smoke density, wildfire information, and participant survey responses on indoor and outdoor PAH air concentrations during wildfires. PAH air concentrations were first summed by ring size for each sample,

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grouped by indoor or outdoor, and then tested as the response variable. A Holm−Bonferroni procedure was used to adjust the significance level and reduce the probability of type I errors.

RESULTS AND DISCUSSION

Impact of Wildfires on Indoor and Outdoor PAH Air Concentrations. We found no differences before, during, or after wildfires when evaluating the sum of all PAHs. However, LMW PAHs were detected at much higher concentrations (3−6 times higher), which dominated the analysis and drowned out the HMW PAHs. As a result, we conducted additional analyses separating out LMW and HMW PAHs.

We found 12 HMW PAHs in outdoor samples during wildfire sampling that were not detected outdoors before or after wildfires (Table 1). To the best of our knowledge, only 4 have been previously reported[51−53] and 8 of the 12 PAHs have not previously been detected in air during wildfires. These 12 PAHs were never detected in indoor samples.

Table 1. Twelve PAHs Were Only Detected during Wildfires in Outdoor Samples[a]

PAH	ring number	number of detections	average AQI of samples
dibenzo[*e*,*l*]pyrene	6-ring	$n = 6$	90, 116, 142, 184, 189, 220
6-methylchrysene	4-ring	$n = 4$	135, 184, 189, 220
7,12-dimethylbenz[*a*]anthracene	5-ring	$n = 4$	135, 184, 189, 220
anthanthrene	6-ring	$n = 4$	116, 142, 189, 220
5-methylchrysene	4-ring	$n = 3$	135, 189, 220
benzo[*a*]chrysene	5-ring	$n = 2$	189, 220
naphtho[2,3-*a*]pyrene	6-ring	$n = 2$	189, 220
naphtho[2,3-*e*]pyrene	6-ring	$n = 2$	189, 220
naphtho[1,2-*b*]fluoranthene	6-ring	$n = 2$	189, 220
coronene	7-ring	$n = 2$	189, 220
perylene	5-ring	$n = 1$	220
dibenzo[*a*,*l*]pyrene	6-ring	$n = 1$	220

[a]Eight of the PAHs (bold) have not been previously reported in air during wildfires.

HMW PAHs, in particular, are an important consideration for exposure due to their potential for increased persistence, bioaccumulation, and toxicity[54] compared to LMW PAHs. Our results suggest that a common high-molecular-weight wildfire PAH exposure mixture could be prioritized for future toxicology and epidemiology studies.[55,56]

Before, during, and after wildfire, PAH comparisons and statistical analysis are presented in Figure S3 and Table 2, respectively. The average indoor (Figure S3a) and outdoor (Figure S3b) vapor-phase LMW PAH air concentrations were three times higher during wildfires than before or after but were not statistically significant (Table 2). Our results are consistent with previous studies on indoor air quality, where the influence of the infiltrating outdoor air was not significant for LMW PAHs.[57,58]

The average vapor-phase HMW PAH air concentrations were six times higher indoors during wildfires than before or after. However, due to nondetect frequency or only $n = 1$ PAH being detected, HMW air concentrations were not statistically significant at most locations (Table 2). Our results are supported by previous studies identifying outdoor air as a contributor to indoor HMW PAHs.[57,58] Specifically, indoor air concentrations of 4-ring PAHs were statistically higher during wildfires at four locations (Table 2). Few 5- and 6-ring PAHs were detected in indoor air. The average outdoor air concentrations of vapor-phase HMW PAHs were 86 times higher during wildfires than before or after and were statistically significant at most locations (Table 2). Concentration increases from our study are slightly higher than other studies,[51,52] though these previous studies have only analyzed for up to 23 PAHs. In our study, we analyzed for up to 65 individual PAHs.

Air concentrations of HMW PAH outdoors also appeared to increase with increasing smoke intensity and AQI values (Figure S3b). Five-ring PAH outdoor air concentrations were significantly higher during wildfires than before or after at seven locations (Table 2). All seven locations had average AQIs exceeding 140 during wildfire sampling (described as unhealthy for sensitive groups). Six-ring PAH air concentrations were significantly higher outdoors at three locations during wildfires than before or after (Table 2). Two of the three locations had the highest average AQI values during wildfire sampling (described as unhealthy and very unhealthy). Similar results have been observed in a previous study focused on bulk particulate matter concentrations.[59]

Indoor versus Outdoor PAH Air Concentrations before, during, and after Wildfires. Indoor minus outdoor (indoor−outdoor) PAH air concentrations for each sampling event are presented in Figure 2.

Before wildfires, indoor vapor-phase PAH air concentrations were significantly higher than outdoor concentrations at all locations except St. Helena, CA (Figure 2a and Table 3). Potential explanations for specific site differences are discussed below. A 2021 review of particulate PAH concentrations in indoor dust samples observed a similar trend, with median sum PAH indoor air concentrations being approximately 2.6 times higher than outdoor air across the globe.[60] Other studies largely focused on PAHs from particulate matter indicate that indoor−outdoor ratios may differ for individual PAHs.[61−63] Personal behaviors and different PAH sources used inside the home have been highlighted as potential causes of different study results.[58,63,64]

Surprisingly, during wildfires, indoor vapor-phase PAH air concentrations were still significantly higher than outdoor concentrations at locations with an average AQI of less than 115, described as good, moderate, or unhealthy for sensitive group air quality (Figure 2b and Table 3). These results are similar to those observed by Messier et al.[24] However, a shift occurred at locations with an average AQI exceeding 115 (unhealthy for sensitive groups), where outdoor HMW PAH air concentrations exceeded indoor concentrations (Figure 2b). Additionally, indoor air concentrations were not significantly higher than outdoor air concentrations (Table 3) with the exception of Sunriver, OR. Potential explanations for specific site differences are discussed below. These results suggest that indoor vapor-phase PAH air quality can be worse than outdoor air quality during mild wildfire conditions.

Prior studies on particulate-bound PAHs have found different results, wherein outdoor air concentrations were consistently higher than indoor air concentrations.[65−67] Since PAHs can be present in both the vapor phase and particulate phase,[19] and vapor-phase PAHs can account for up to 86% of the cancer risk from total inhalation exposure (vapor phase and particulate matter),[20−23] our results provide an important

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Table 2. One-Sided Paired *t*-Test Comparing Indoor and Outdoor PAH Air Concentrations before, during, and after Wildfires by Ring Size[a]

		LMW PAHs		HMW PAHs			
	location	2-ring	3-ring	4-ring	5-ring	6-ring	wildfire average AQI
before wildfires indoor[b]	Alturas, CA	0.99	0.90	0.10	0.10	0.10	70
	Newport, OR	0.34	0.99	0.83	N/A (n = 0)	N/A (n = 0)	90
	St. Helena, CA	**<0.00010**[b]	**0.0069**[b]	**0.00020**[b]	N/A (n = 0)	N/A (n = 0)	93
	Sandpoint, ID	0.24	0.58	0.57	N/A (n = 0)	N/A (n = 0)	116
	Richland, WA	0.0088	0.25	**<0.00010**[b]	0.012	N/A (n = 1)	135
	Lake Oswego, OR	0.019	0.22	0.014	0.038	N/A (n = 1)	189
after wildfires indoor[c]	Carson, WA	**<0.00010**[c]	0.88	0.16	N/A (n = 0)	N/A (n = 0)	21
	McCall, ID	0.36	0.26	0.044	N/A (n = 0)	N/A (n = 0)	73
	Prineville, OR	0.59	0.92	0.090	N/A (n = 1)	N/A (n = 0)	95
	Sandpoint, ID	0.096	0.24	0.74	N/A (n = 0)	N/A (n = 0)	116
	Richland, WA	0.018	0.54	**<0.0001**[c]	0.012	N/A (n = 1)	135
	Seattle, WA 2	0.85	0.91	0.011	N/A (n = 0)	N/A (n = 1)	142
	Sunriver, OR	0.14	0.98	**0.0050**[c]	N/A (n = 1)	N/A (n = 0)	184
	Lake Oswego, OR	0.085	**0.0030**[c]	**0.00030**[c]	0.015	N/A (n = 1)	189
	Corvallis, OR 2	0.10	0.93	0.0086	0.032	0.041	220
before wildfires outdoor[b]	Alturas, CA	0.92	0.95	1.0	1.0	1.0	70
	Newport, OR	0.79	0.16	**0.00050**[b]	N/A (n = 0)	N/A (n = 1)	90
	St. Helena, CA	0.033	0.025	**<0.00010**[b]	0.016	N/A (n = 1)	93
	Sandpoint, ID	0.50	0.053	**<0.00010**[b]	**0.0032**[b]	**0.0049**[b]	116
	Richland, WA	0.061	0.033	**<0.00010**[b]	**0.0002**[b]	N/A (n = 1)	135
	Lake Oswego, OR	0.14	**0.0054**[b]	**<0.00010**[b]	**<0.00010**[b]	**<0.00010**[b]	189
after wildfires outdoor[c]	Carson, WA	0.99	0.80	0.99	0.99	N/A (n = 0)	21
	McCall, ID	**0.00020**[c]	0.012	**<0.00010**[c]	N/A (n = 0)	N/A (n = 0)	73
	Prineville, OR	0.0099	0.051	**<0.00010**[c]	**0.00080**[c]	0.044	95
	Sandpoint, ID	0.94	0.016	**<0.00010**[c]	**0.00010**[c]	**<0.00010**[c]	116
	Richland, WA	0.99	0.011	**<0.00010**[c]	**0.00030**[c]	N/A (n = 1)	135
	Seattle, WA 2	**<0.0001**[c]	**0.0020**[c]	**<0.00010**[c]	0.016	0.049	142
	Sunriver, OR	0.19	0.92	1.0	1.0	0.97	184
	Lake Oswego, OR	0.48	0.011	**<0.00010**[c]	**<0.00010**[c]	**<0.00010**[c]	189
	Corvallis, OR 2	0.61	**0.00020**[c]	**<0.00010**[c]	**<0.00010**[c]	**<0.00010**[c]	220

[a]Significant p-values indicate that vapor-phase PAH air concentrations during wildfires are significantly greater than before or after. α values are Bonferroni-corrected for each sampling event. [b]Significant when probability >t at α' = 0.0083. [c]Significant when probability >t at α' = 0.0056.

consideration for public health recommendations during wildfire events.

Previous studies have also focused on air infiltration indoors,[65−67] and often excluded or subtracted out indoor sources of particulate matter, in attempts to only capture PAH air concentrations related to wildfire smoke infiltration.[65] In this approach, PAH concentrations from outdoor air infiltration alone do not capture the total indoor air quality, where residents are being exposed to sources from inside and outside the home. Therefore, the concentrations presented in this study represent a more accurate measure of human exposure indoors during wildfires.

To evaluate the potential long-term effects of extreme wildfire smoke events, we compared indoor−outdoor vapor-phase PAH air concentrations after wildfires in 2018 and 2020. Following mild smoke impact (2018), indoor vapor-phase PAH air concentrations were significantly higher than outdoor air concentrations (Figure 2c and Table 2). In contrast, 2020 indoor air concentrations for three of the four locations (Seattle, WA, Sunriver, OR, and Corvallis, OR) were not significantly higher than outdoor air concentrations (Table 2). These results suggest that outdoor air could still be impacted by HMW vapor-phase PAHs after extreme wildfire events.

Influence of Wildfire Characteristics and Participant Behaviors on Indoor and Outdoor Air PAH Concentrations. HMW PAH concentrations in indoor and outdoor air during wildfires are plotted against the average AQI in Figure 3. A significant (p < 0.010) linear relationship was found between the average AQI and outdoor air concentrations of HMW PAHs during wildfires (Figure 3) but not indoor air. Figure 3 also highlights that below an AQI of 140, indoor HMW PAH air concentrations are higher than outdoor air concentrations. Above an AQI of 140, a shift occurs, and outdoor HMW PAH air concentrations are higher than indoor air concentrations. No significant relationship was found for LMW PAH air concentrations and average AQI.

No significant relationships were found between air concentrations and NOAA smoke density or any wildfire parameters (wildfire size, distance from wildfire, etc.). Future research should explore other parameters for their influence on PAH concentrations during wildfires.

Survey responses and comments from participants were examined to identify the potential influence of human behavior on PAH air concentrations indoors and outdoors. No significant relationships were found between indoor air and participant survey responses. A lack of significant correlations could be due to incomplete survey data, with compliance at 55% for 2019 and 2020.

Some locations, such as Alturas, CA, and Sunriver, OR, did not follow the expected trends when comparing PAH air

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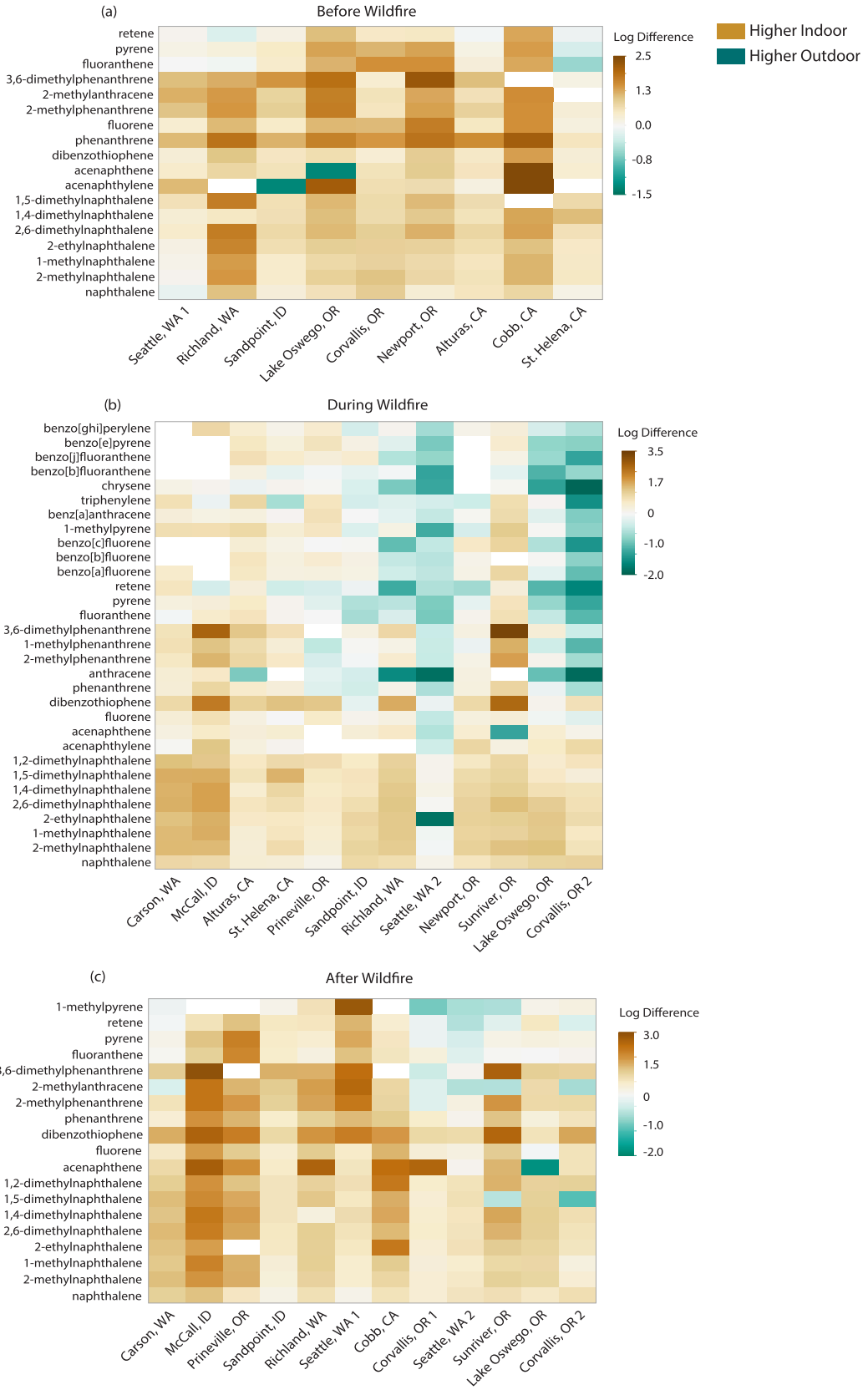

Figure 2. Heat map of indoor−outdoor vapor-phase PAH concentrations (a) before, (b) during, and (c) after wildfires. Air concentrations were first log-transformed and then subtracted. Only PAHs detected in at least 75% of samples are included. Shades of yellow represent higher PAH concentrations indoors, while shades of blue represent higher PAH air concentrations outdoors.

concentrations before, during, and after wildfires. Alturas, CA, had similar PAH air concentrations indoors before and during wildfires. Sunriver, OR, had higher PAH air concentrations outdoors after wildfires than during wildfires. Survey responses

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Table 3. One-Sided Paired *t*-Test Comparing Indoor−Outdoor PAH Air Concentrations before, during, and after Wildfires[a]

location	before wildfire[b]	wildfire[c]	after wildfire[d]	wildfire average AQI
Seattle, WA 1	0.0025*	N/A	<0.0001*	N/A
Cobb, CA	<0.00010*	N/A	<0.0001*	N/A
Corvallis, OR 1	<0.00010*	N/A	0.0290[e]	N/A
Carson, WA	N/A	<0.00010*	<0.00010*	21
Newport, OR	<0.00010*	<0.00010*	N/A	70
McCall, ID	N/A	<0.00010*	<0.00010*	73
Alturas, CA	<0.00010*	<0.00010*	N/A	90
St. Helena, CA	0.043	<0.00010*	N/A	93
Prineville, OR	N/A	<0.00010*	<0.00010*	95
Sandpoint, ID	<0.00010*	0.473	<0.00010*	116
Richland, WA	0.0010*	0.15	<0.00010*	135
Seattle, WA 2	N/A	1.0	0.76	142
Sunriver, OR	N/A	<0.00010*	0.057	184
Lake Oswego, OR	<0.00010*	0.69	0.0015*	189
Corvallis, OR 2	N/A	1.0	0.18	220

[a]Significant *p*-values indicate that vapor-phase PAH air concentrations indoors are significantly greater than those outdoors. α values are Bonferroni-corrected for each sampling event. [b]*Significant when probability <*t* at $\alpha' = 0.0056$. [c]*Significant when probability <*t* at $\alpha' = 0.0042$. [d]*Significant when probability <*t* at $\alpha' = 0.0042$. [e]Potential impact due to backyard campfire near the outdoor air sampler.

indicated that the participants themselves owned a woodstove that was used when sampling before/after wildfires, and woodstoves were also being used by other residents in the area. Previous studies have noted the impact of woodstove heating as a significant source of PAH air concentrations.[68,69] Therefore, woodstove use could explain higher PAH concentrations in these Alturas and Sunriver samples.

Additionally, Sunriver, OR, and St. Helena, CA, did not follow the trends seen for other locations when comparing indoor and outdoor PAH air concentrations. Sunriver, OR, had significantly higher indoor PAH air concentrations during wildfires, even though this location had an average AQI of 189. St. Helena, CA, had similar indoor and outdoor concentrations before wildfires. Survey responses did not indicate any personal behaviors that would account for higher indoor concentrations. Home age, ventilation, and other conditions could account for these differences.[70,71] Another contributing factor could be the distance of the AQI monitor from the sampling location and/or prevailing wind direction of smoke in relation to the AQI monitor. The closest U.S. EPA AQI monitor was 17 miles away for Sunriver, OR, and 20 miles away for St. Helena, CA (Tables S6 and S7).

Survey responses were also examined for any potential changes in personal behaviors during each sampling event. On average, participants indicated that when sampling before wildfires, windows were opened for 10 (range: 0−44) days (Table S13). However, on average, participants only opened windows for 2 (range: 0−6) days during wildfires and 5 (range: 0−21) days after wildfires (Table S13). No changes in behavior were observed with air conditioning use. Other studies have shown the large role of personal behaviors on indoor air exposure, in addition to household characteristics.[72] Future research should further explore the effects of personal and household variables on indoor PAH exposure under various wildfire conditions.

Exploratory Assessment of Wildfire Influence on Cancer and Noncancer Inhalation Risks. Cancer risk and noncancer hazard values were compared before, during, and after wildfires for indoor and outdoor PAH inhalation exposures. Indoors and outdoors, 80% of locations had a higher inhalation cancer risk and hazard quotient during wildfires than before or after (Tables S18 and S19). Indoor inhalation risk (cancer and noncancer) was three times higher during wildfires, while outdoor inhalation risk was 36 times

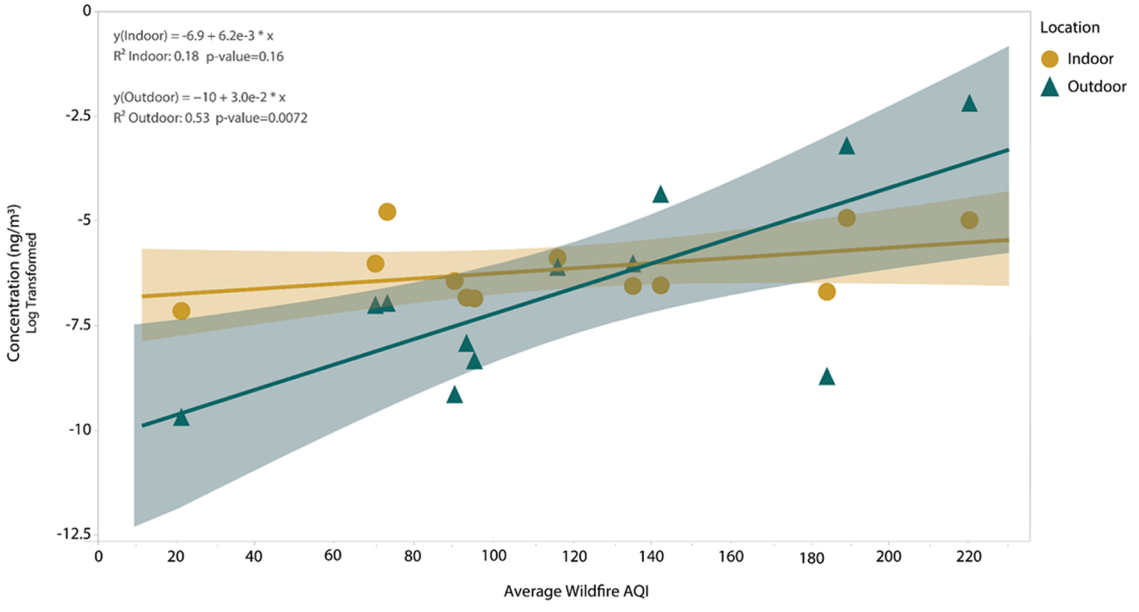

Figure 3. Linear regression models during wildfires for sum HMW PAH air concentrations (log-transformed) and average AQI. Air concentrations for indoor (yellow circles) and outdoor (blue triangles) samples are each plotted against the average AQI at all locations. The α value is Holm−Bonferroni-corrected at $\alpha' = 0.010$.

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Table 4. Inhalation Cancer Risk and Noncancer Hazard Indoors Compared to Outdoors during Wildfires[a]

location	indoor cancer risk	outdoor cancer risk	indoor hazard quotient	outdoor hazard quotient	wildfire average AQI
Carson, WA	**3.0×10^{-7}**	1.2×10^{-8}	**3.5×10^{-3}**	3.4×10^{-5}	21
Newport, OR	**6.1×10^{-8}**	4.8×10^{-9}	**2.5×10^{-4}**	5.2×10^{-6}	70
McCall, ID	**4.0×10^{-7}**	1.1×10^{-8}	**2.1×10^{-3}**	2.4×10^{-5}	73
Alturas, CA	**5.2×10^{-7}**	1.5×10^{-8}	**1.6×10^{-3}**	6.2×10^{-5}	90
St. Helena, CA	**2.9×10^{-7}**	2.4×10^{-8}	**7.6×10^{-4}**	3.2×10^{-5}	93
Prineville, OR	**9.9×10^{-8}**	1.2×10^{-8}	**4.3×10^{-4}**	3.8×10^{-5}	95
Sandpoint, ID	**3.5×10^{-7}**	5.1×10^{-8}	**3.2×10^{-3}**	4.6×10^{-4}	116
Richland, WA	**3.7×10^{-7}**	4.6×10^{-8}	**2.2×10^{-3}**	3.2×10^{-5}	135
Seattle, WA 2	**1.6×10^{-7}**	1.1×10^{-7}	**7.1×10^{-4}**	6.9×10^{-5}	142
Sunriver, OR	**2.5×10^{-7}**	5.6×10^{-9}	**5.9×10^{-4}**	7.9×10^{-6}	184
Lake Oswego, OR	**7.6×10^{-7}**	2.0×10^{-7}	**1.2×10^{-3}**	3.2×10^{-5}	189
Corvallis, OR 2	**7.6×10^{-7}**	4.6×10^{-7}	**7.7×10^{-4}**	6.5×10^{-5}	220

[a]Values in bold indicate the higher risk for a particular site.

higher. Our study results suggest that human inhalation exposure to PAHs from wildfire smoke increases both cancer and noncancer risks, regardless of indoor or outdoor location, even over a short period of exposure. Messier et al.[24] did not previously observe significant changes in cancer risk with increased smoke impact but only sampled during mild wildfire conditions.

Cancer risk and noncancer hazard values were also compared for indoor versus outdoor PAH inhalation exposure during wildfires. All 12 locations had a higher risk indoors than outdoors during wildfires (Table 4). Similar results were found in Messier et al. during wildfire sampling using a toxicity equivalency approach.[24] Cancer risk was largely driven by naphthalene, fluoranthene, 7,12-dimethylbenz[*a*]anthracene, and benzo[*j*]fluoranthene, while noncancer hazard was largely driven by naphthalene. A few other studies[24,73−76] have compared inhalation risk to PAHs in indoor versus outdoor air. Of the studies conducted, the focus has been primarily on particulate matter and found that risk to outdoor air is higher than indoor air.[73−76] However, other recent studies have found that indoor cancer risk can be higher than or similar to risk from outdoor air.[77,78]

Future studies should examine the risk of vapor-phase PAHs in indoor and outdoor air irrespective of wildfires to provide a more comprehensive assessment of PAH risk in both environments.

Recommendations. Results from our study suggest that current public health recommendations during wildfires, relying mostly on outdoor air quality and particulate matter, may not be sufficient for reducing human PAH exposure. Improved recommendations may highlight not only keeping smoke out of the home but also keeping indoor air clean from other vapor-phase and particulate PAH sources through increased ventilation and use of higher efficiency filters, which can remove both particulate and vapor-phase contaminants. Additionally, more protective recommendations may be needed for sensitive populations.

Due to incomplete survey responses, no significant relationships were found between indoor PAH air concentrations and other indoor PAH sources. However, our results indicate that there are ongoing indoor sources of PAHs, which contribute to high indoor concentrations. Indoor sources of vapor-phase PAHs should be explored further in a future study.

There are limitations to wildfire sampling studies using stationary monitors. Previous studies have used outdoor PAH air concentrations from public stationary monitoring systems rather than pairing an outdoor sampler at each sampling site.[65−67] Distance from the monitor to the home could lead to a loss in the resolution of exposure, as seen in our study, and depending on the prevailing wind direction may not be an accurate reflection of local air quality (e.g., monitor may be upwind of smoke). However, by codeploying passive samplers at the same residential location in our study, a better representation of true indoor and outdoor PAH exposures was measured.

An additional limitation is that the PAH inhalation risk assessments provided in this study represent only a fraction of the entire PAH mixture. Without a comprehensive list of reference values for all PAHs, it is difficult to determine the true risks associated with indoor and outdoor exposures. Further research into additional PAH compounds and mixtures is needed to provide a more realistic cancer risk and hazard quotient estimate.

■ ASSOCIATED CONTENT

Supporting Information

The Supporting Information is available free of charge at https://pubs.acs.org/doi/10.1021/acs.est.2c00619.

The Supporting Information contains additional details about sample preparation, collected metadata, analytical methodology, calculations, environmental concentrations, and data analysis (PDF)

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Notes

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