Firefighters’ exposures to combustion-derived polycyclic aromatic hydrocarbons and other mutagens

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Abstract

The objective of this thesis was to investigate firefighters' exposures to carcinogens and mutagens. This thesis aimed to (1) characterize firefighters' exposures during emergency fire suppression, (2) examine the use of silicone wristbands as passive samplers to assess firefighters' exposures, and (3) assess the ability of post-fire decontamination protocols to reduce firefighters' exposures. Chapter 1 provides a general introduction and background information on the concepts covered in this thesis. In Chapter 2, I examined air and surface contamination with polycyclic aromatic hydrocarbons (PAHs) and metals, both at fire stations and following emergency fire suppression activities. I also investigated the ability of current laundering methods to remove surficial PAHs and metals from firefighters' personal protective equipment. In Chapter 3, I assessed the ability of silicone wristband passive samplers to measure firefighters' exposures to PAHs during live fire training. In Chapter 4, I assessed the ability of three post-fire dermal decontamination methods to remove surface contamination and reduce internal dose. In Chapter 5, conclusions and implications for each chapter are summarized. Future directions for the field are described. The overall conclusions for this thesis were: i. firefighters experience significant occupational exposures to carcinogens during emergency fire suppression and live fire training; ii. the ability of silicone wristbands to properly sample PAHs is altered in a fire environment and correction factors must be determined to improve their utility for exposure assessment; iii. current post-fire dermal decontamination methods, which are intended to reduce firefighters' exposures, do not reduce their internal dose of PAHs.

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Acknowledgments

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My grandmothers knew more about my research than most. When in doubt, I often think of Carol telling me to “give ‘em hell”. Doris, while in her 90s, was discussing my research with her nurses and even set up an opportunity for me to share my research with a nearby fire department in Alberta. Although I lost both during my PhD, I will forever be thankful to have had my grandmothers’ encouragement and support.

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Last, and most importantly, to the loves of my life, Jo and Lauriann: thank you for always lighting up my world. Je vous aime.

List of Abbreviations

1-OHP	1-Hydroxypyrene
ACGIH	American Conference of Governmental Industrial Hygienists
AKR	Aldo-keto reductases
AM	Arithmetic mean
ANOVA	Analysis of variance
aOR	Adjusted odds ratio
ASE	Accelerated solvent extraction
BaP	Benzo(a)pyrene
Cd	Cadmium
FRs	Flame retardants
GM	Geometric mean
HMW	High molecular weight
HPLC	High performance liquid chromatography
HR	Hazard ratio
HVAS	High volume air sample
IARC	International Agency for Research on Cancer
LANSET	Laboratory for the Analysis of Natural and Synthetic Environmental Toxicants (University of Ottawa)
LMW	Low molecular weight
MSD	Mass selective detector
NIOSH	National Institute of Occupational Health and Safety
NIST	National Institute of Standards and Technology
NSERC CREATE-REACT	Natural Sciences and Engineering Research Council - Collaborative Research and Training Experience Program - Research in Environmental, Analytical, and Chemical Toxicology
OEL	Occupational exposure limit
OFS	Ottawa Fire Services
OPFR	Organophosphate flame retardants
OSHA	Occupational Safety and Health Administration
PAH	Polycyclic aromatic hydrocarbon
PAS	Personal air sample
Pb	Lead
PBDE	Polybrominated diphenyl ethers
PFAS	Per- and poly-fluorinated alkyl substances
PPE	Personal protective equipment
PUF	Polyurethane foam
Sb	Antimony
SCBA	Self-contained breathing apparatus
SC	Stratum corneum
SE	Standard error
SMR	Standardised mortality ratio
SPE	Solid phase extraction
SWB	Silicone wristband

TWA	Time weighted average
USEPA	United States Environmental Protection Agency
WPF	Worker protection factor

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Preface

This thesis was written by manuscript in accordance with the guidelines provided by the University of Ottawa's Faculty of Graduate and Postdoctoral Studies. Chapter 1 provides an introduction and background to the thesis and its manuscripts. Chapter 2 is a manuscript formatted to Elsevier's guidelines for authors provided by the journal *Science of the Total Environment*. Chapter 3 is a manuscript formatted according to the guidelines for authors provided by the Journal, *Environment International*. Chapter 4 is a manuscript formatted to the *Journal of Occupational and Environmental Hygiene*. Chapter 5 provides a summary, concluding remarks, and future directions for this field.

Chapter 1 Introduction

Firefighters' health risks

It is often thought that the main risks firefighters face while on the job are immediate hazards such as heat or building collapse. However, the leading cause of on-duty death for firefighters, comprising 45% of on-duty fatalities, is cardiovascular disease, and the overall leading cause of death for firefighters is cancer (Centers for Disease Control and Prevention 2006). In an analysis of data from 2009-2018, 89.7% of firefighter fatality claims in Canada were reportedly related to cancer, versus just 4.9% for traumatic injury (Thomas, Garis, and Biantoro 2020). A large-scale study out of the United States of nearly 30,000 firefighters found that firefighters have a 9% higher risk of developing cancer and 14% higher risk of dying of cancer compared to the general population (Daniels et al. 2014). A more recent study of 100,000 career Florida firefighters, including over 5,000 female firefighters, calculated gender-specific age and calendar year-adjusted odds ratios (aOR) and 95% confidence intervals for firefighters versus non-firefighters. Female firefighters had significantly elevated risk of brain (aOR=2.54; 1.19-5.42) and thyroid (2.42; 1.56-3.74) cancers. Male firefighters were found to be at an increased risk of melanoma (1.56; 1.39-1.76), prostate (1.36; 1.27-1.46), testicular (1.66; 1.34-2.06), thyroid (2.17; 1.78-2.66) and late-stage colon cancer (1.19;1.00-1.41) (Lee et al. 2020). More locally, Sritharan et al. (2022) identified a cohort of 13,642 Ontario firefighters who had cancer between 1983 and 2020; they found that compared with other workers, firefighters had increased risk of prostate cancer (HR=1.43, 95% CI 1.31 to 1.57), colon cancer (HR=1.39, 95% CI 1.19 to 1.63) and skin melanoma (HR=2.38, 95% CI 1.99 to 2.84); police also had increased risks for these cancers (Sritharan et al. 2022).

In addition, firefighters also had increased risk of cancer of the pancreas, testis, and kidney, and non-Hodgkin's lymphoma and leukaemia. On an international scale, the International Agency for Research on Cancer (IARC) recently re-evaluated the carcinogenicity of occupational exposure as a firefighter and reclassified firefighting as a Group 1 known human carcinogen (Demers et al. 2022).

There are a variety of cancer risk factors that firefighters may experience including stress, disruptions in circadian rhythm, obesity, smokeless tobacco use, and alcohol abuse (Gulliver et al. 2019; Jitnarin et al. 2015; Lim et al. 2020; Reinberg et al. 2017). Exposure to carcinogenic and mutagenic combustion by-products may also contribute to firefighters' cancer risk. In fact, a variety of toxic chemicals have been measured in combustion emissions at firefighting events including metals (e.g., cadmium, antimony, lead), particles and fibers (e.g., asbestos, silica dust) (Austin et al. 2010; Guidotti 2016), and a wide range of organic substances. The latter includes numerous carcinogenic and mutagenic substances such as benzo[*a*]pyrene, benzene, styrene, acrolein, formaldehyde, 1,3-butadiene, phenols, dioxins (Kirk and Logan 2015; Lönnermark and Blomqvist 2006; Naeher et al. 2007; De Vos et al. 2009). When discussing firefighters' higher rates of cancer and illness compared to the general population, it is important to consider exposures to toxic substances.

Metals

Several metals, such as cadmium, antimony, and lead, have been detected from firefighter activities, both in environmental air during fire suppression and on firefighter surfaces afterwards (e.g., gloves) (Bolstad-Johnson et al., 2000; Guidotti, 2015). Cadmium has been classified by IARC as a Group 1 carcinogen; antimony trioxide and lead have both been classified as Group 2B, i.e., possibly carcinogenic to humans (IARC, 1989, 1993, 2006). Exposures to these compounds can occur via dermal, oral, or respiratory routes. In the context of firefighters' exposures, the main routes of exposure are thought to be respiratory and dermal Past studies have shown all three metals present in air and on surfaces of firefighters' equipment (Fabian et al., 2014); they have been shown to be dermally absorbed in animal and/or human studies (Wang et al., 2021, Collin et al., 2022, Tylenda et al., 2022). Metabolism and excretion of metals are generally slow, with cadmium having a biological half life of 10 to 30 years, followed by lead at 30 days, and antimony at ~4 days (Wang et al., 2021, Collin et al., 2022, Tylenda et al., 2022). Current literature is limited on information on metals in firefighters' occupational environmentswhile on-shift at the fire station and during emergency fire suppression.

Polycyclic Aromatic Hydrocarbons (PAHs)

Hundreds of chemicals are produced during combustion, and combustion of both natural and anthropogenic materials can release hazardous chemicals into the environment. PAHs were the focus of this thesis, as they are noteworthy carcinogens and may influence firefighters' cancer risks.

PAHs are a group of organic compounds formed during combustion of organic matter; they are commonly found in barbequed or smoked foods, wildland fire emissions, bonfire emissions, and tobacco smoke. They are ubiquitous and some have carcinogenic, mutagenic, and teratogenic properties. Of the hundreds of PAHs, 16 were selected in 1976 by the US Environmental Protection Agency (US EPA) as priorities for concern and control (Keith and Telliard 1979). Their structures are illustrated in Figure 1-1. Importantly, the International Agency for Research on Cancer (IARC) has classified certain PAHs as either known, probable, or possible carcinogens. Specifically, benzo[*a*]pyrene (BaP) is currently classified as a known human carcinogen (Group 1). Dibenz[*a,h*]anthracene and dibenzo[*a,l*]pyrene are classified as probable human carcinogens (Group 2A), and benz[*j*]aceanthrylene, benz[*a*]anthracene, benzo[*b*]fluoranthene, benzo[*j*]fluoranthene, benzo[*k*]fluoranthene, benzo[*c*]phenanthrene, chrysene, dibenzo[*a,h*]pyrene, dibenzo[*a,i*]pyrene, indeno[1,2,3-*cd*]pyrene and 5-methylchrysene are classified as possible human carcinogens (Group 2B) (IARC 2010). As such, release into the environment, and human exposure to these and other PAHs, requires attention.

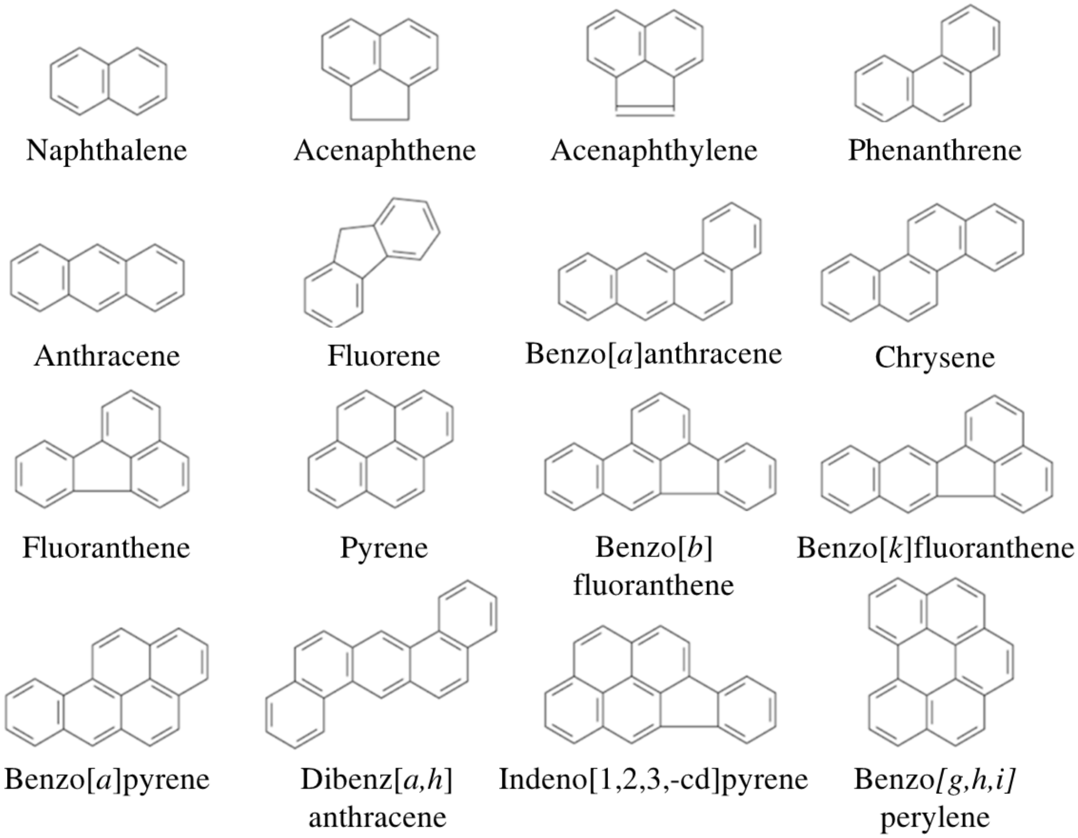

Figure 1-1 Structures of the 16 priority PAHs.

PAHs span a variety of physical and chemical properties. Table 1-1 lists some of the physical properties of the 16 priority PAHs. Although PAHs are grouped together, they can differ significantly in their origin and physical-chemical characteristics. In general, low molecular weight (MW) PAHs are more abundant in environmental samples and tend to be more volatile, with naphthalene being notoriously difficult to measure due to its volatility. In contrast, high molecular weight (HMW) PAHs tend to be found on particles. The formation of various PAHs is dependant on both temperature and fuel. For example, Banks et al. (2021) found the concentrations of all measured PAHs to be significantly higher from particleboard fires compared to diesel pan fires ($p < 0.05$). Han et al. (2020) found that the formation of high MW PAHs during wood combustion increased with increasing temperature; formation decreased during coal combustion.

Table 1-1 Summary of physical-chemical properties of the 16 priority PAHs at 298 K (25°C). (data from Hansch, Leo, and Hoekman 1995; Haynes 2015; Ma et al. 2010)

	Molecular Weight (g mol^{-1})	**# of rings**	**Melting Point (K)**	**Log K_{ow}**	**Log K_{OA}**
Naphthalene	128.17	2	353.1	3.4	5.19
Acenaphthylene	152.19	3	365.1	3.85	6.46
Acenaphthene	154.21	3	366.3	3.95	6.44
Fluorene	166.22	3	387.7	4.11	6.85
Phenanthrene	178.23	3	372	4.47	7.64
Anthracene	178.23	3	489.8	4.57	7.7
Pyrene	202.25	4	423.4	5.01	8.86
Fluoranthene	202.25	4	382	4.97	8.81
Chrysene	228.29	4	530	5.67	10.3
Benzo[*a*]anthracene	228.29	4	431.8	5.83	10.28
Benzo[*b*]fluoranthene	252.31	5	441.2	5.86	11.3
Benzo[*k*]fluoranthene	252.31	5	485.4	5.86	11.36
Benzo[*a*]pyrene	252.31	5	452	6.05	11.48
Benzo[*g,h,i*]perylene	276.33	6	547.7	6.63	12.55
Indeno[1,2,3-cd]pyrene	276.33	6	436	6.57	12.43
Dibenz[a,h]anthracene	278.35	5	516.2	6.5	NA

NA= not available

PAH exposures

PAHs may enter the body via dermal, respiratory, or oral routes. For a typical non-smoking, non-occupationally exposed individual, ingestion of PAHs is thought to be the main route of exposure (Ramesh et al. 2004). For firefighters, however, their exposures are unique because of the extreme environments they are exposed to and their personal protective equipment. Dermal contact and inhalation are thought to be the main routes of firefighters' PAH exposures.

Dermal PAH exposures

The skin is the largest organ of the human body, composed of the epidermis, dermis and hypodermis (Figure 1-2). The epidermis is the outermost layer, made of mainly keratinocytes; it is the primary barrier against loss of salt and water, and entry of foreign materials (e.g., chemicals, pathogens). The epidermis can be further divided into four layers according to the differentiation of keratinocytes into horny cells, including the outermost horny cell layer (stratum corneum), followed by the granular cell layer (statrum granulosum), squamous cell layer (stratum spinosum), and the basal cell layer (stratum germinativum) (Kolarsick, Kolarsick, and Goodwin 2011). Beneath the epidermis is the dermis, containing hair follicles and sweat glands, and comprised of fibroblasts that synthesize collagen and elastin fibres. Below the dermis is the hypodermis, and an area comprised mainly of fatty acids. The hypodermis connects the dermis layer to muscles and bones, and provides thermoregulation, insulation, stores nutrition, and protects deeper tissues from injury. The hypodermis contains veins and arteries that branch into arterioles and venules, and ultimately capillaries, in the dermis layer (Kanitakis 2002; Venus, Waterman, and McNab 2010). Environmental contaminants deposited on the skin are dissolved in the skin-surface film liquid (SSFL) on the epidermis (Pawar et al. 2016).

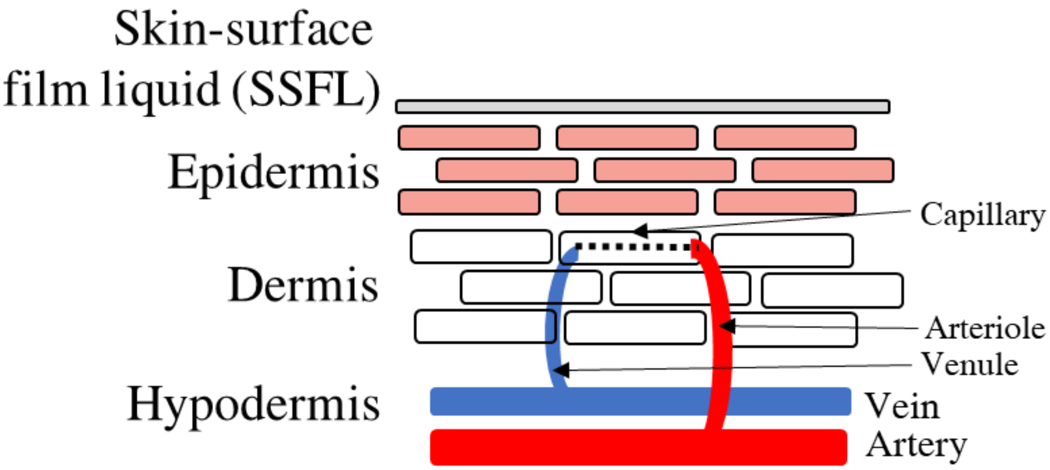

Figure 1-2 Layers of human skin, adapted from Pawar et al. (2016)

There are several steps involved in dermal absorption of PAHs. First, for larger molecular weight PAHs that are attached to particulate matter (PM), the PM undergoes dissolution, releasing the PAH from the particle. PAHs partition out of the vehicle and into the stratum corneum (SC) of the epidermis. Substances are thought to enter the SC via passive diffusion through the intercellular spaces (Hadgraft 2004). After the SC, the remaining layers of the epidermis and dermis, which contain circulatory system components, become barriers controlling the flux of substances into systemic circulation (Yamaguchi et al. 2008).

The absorption of PAHs into the SC is dependent on physicochemical characteristics including lipophilicity, molecular size, and vehicle type (Alalaiwe et al. 2020). Because of the lipophilic environment of the SC, PAHs of higher lipophilicity have higher partitioning rates into the SC and can even form a reservoir (Bourgart et al. 2019). In fact, one study found that 80% of BaP applied to a skin model remained in the SC and only the remaining 20% penetrated into the other epidermal layers and the receptor fluid of the model (Bartsch et al. 2016). Whereas high lipophilicity aided in penetration of the SC, the remaining layers of skin have hydrophilic properties making it difficult for lipophilic compounds to diffuse into to the lower layers (Alalaiwe et al. 2020; Roy et al. 2006). Yamaguchi et al. (2008) also found skin thickness to influence permeation of highly lipophilic compounds into lower skin layers, with thicker skin having slower penetration rates. Molecular weight has also been shown to have an inverse relationship with the dermal diffusion rates of PAHs (Alalaiwe et al. 2020; Vanrooij et al. 1995; Yamaguchi et al. 2008). Relatedly, structure-activity relationship

(SAR) models have found that PAH log octanol/water partition coefficient (log P) is the most important factor determining skin absorption, with an inverse relationship between log *P* and the skin penetration properties (Roy et al. 2006). Vehicle can also affect the absorption of PAHs (Bartsch et al. 2016; Sartorelli et al. 1999, 2001). For example, several studies have found little percutaneous penetration of PAHs from solids (e.g., coal dust) compared to solutions (e.g., artificial sweat, acetone solutions) (Foa et al. 1998; Sartorelli et al. 2001). All of these factors contribute to the dermal absorption rates of PAHs, which have been studied using a variety of vehicles (e.g., dust, simulated sweat, solvents) (Bartsch et al. 2016; Luo et al. 2020; Payan et al. 2008; Sartorelli et al. 1999). For example, Sartorelli et al. (1999) reported steady state fluxes of 1.0 nmol cm^2 h^{-1} for naphthalene to 0.0014 for nmol cm^2 h^{-1} for benzo[*a*]pyrene. In a firefighter context, high temperatures and volume of sweat may influence penetration behaviour, and ultimately, bioavailability of dermally deposited PAHs.

Metabolism of dermally exposed PAHs can occur both in the skin and elsewhere following systemic circulation. Epidermal keratinocytes have shown metabolism capabilities, resulting in BaP derived genotoxicity (Brinkmann et al. 2013; Theall, Eisinger, and Grunberger 1981; Toshio Kuroki et al. 1982). However, the expression and activity of enzymes involved in PAH metabolism (i.e., CYPs or cytochrome P450 isozymes) are much higher in the liver (Kazem, Linssen, and Gibbs 2019). Thus, PAHs that reach systemic circulation are more likely to be fully metabolised and manifest their toxic effects.

Respiratory PAH exposure

PAHs can also enter the body via the respiratory tract. Upon entering the lungs, either airborne or adsorbed to particles, PAHs can be metabolised by Phase I and II enzymes or be transported to systemic circulation where they are later metabolized (Gerde et al. 2001; Ifegwu and Anyakora 2015; Moorthy, Chu, and Carlin 2015). PAHs are absorbed through the lungs by transport across the mucus layer lining the bronchi and through passive diffusion in the alveoli, partitioning into lipids and aqueous layers of cells (Bevan and Ulman 1991; Gerde, Medinsky, and Bond 1991). Absorption rate of PAHs from the lungs, and entry into systemic circulation, is dependent on the location within the respiratory tract, with slow airway clearance from slower diffusion through the thicker air/blood barrier of the conducting airways compared to fast diffusion through the thinner alveolar epithelium (Gerde et al. 2001; Gerde, Muggenburg, and Henderson 1993). Studies have shown that adsorption of PAHs onto particles affects their retention time in the lung (Sun et al. 1984). Exposure to PAH-containing mixtures has been linked to lung cancer in tobacco smokers and from occupational exposures in coke ovens, gasworks, aluminum production, asphalt, and chimney sweeps (Armstrong et al. 2004; Grimmer et al. 1988), likely as a result of the lung's ability to metabolise PAHs, and consequently generate DNA reactive metabolites.

Air concentrations of PAHs in an individual's environment are often measured using personal air samplers. These samplers generally consist of a pump that draws air through a sample cartridge that includes a filter to collect particle-bound PAHs and a polyurethane foam (PUF) plug to collect volatile compounds. The volume of air collected is recorded by the sampling pump so that concentrations can be reported as mass per sampled volume of air. Personal air sampling for PAHs has been used in a variety of scenarios such as seafarers (Strandberg et al., 2022), pregnant women living in urban, industrial areas (Nethery et al., 2012), and firefighters during live fire training (Banks et al. 2020; Caban-Martinez et al. 2020; Oliveira et al. 2017). Although personal air samplers are useful for measuring overall airborne concentrations of compounds like PAHs, they only provide time-averaged concentrations. Thus they lack the ability to provide insight regarding dynamic changes in concentration over time.

PAH metabolism

PAHs must first enter the body and become metabolized before their toxicity is manifested. Parent PAHs are not carcinogenic or mutagenic, but their metabolites can react with DNA and induce the formation of mutations that can initiate cancer (IARC 2010). PAHs are non-polar and hydrophobic, thus to be eliminated by the body, they must first be transformed into more polar, water-soluble compounds. Figure 1-3 illustrates the various pathways the carcinogenic PAH, benzo[*a*]pyrene (BaP), can be metabolised. Phase I and II detoxification enzymes transform PAHs into hydroxy-PAHs via a variety of pathways; subsequently, they can be conjugated to an endogenous metabolite and eliminated from the body via the urine or faeces. However, Phase I metabolism produces several potent electrophilic intermediates, including epoxides, phenols, quinones, hydroquinones, dihydrodiols, phenol dihydrodiols, dihydrodiol epoxides, and tetrols. These electrophiles can readily react with DNA to form bulky lesions known as adducts, which if unrepaired, can lead to mutations that can contribute to cancer initiation. Figure 1-4 presents an example of a pathway, and resulting structures, of metabolism of BaP leading to a guanine adduct. Due to the metabolism-induced toxicity associated with DNA-reactive intermediates generated prior to excretion, PAHs are sometimes referred to as procarcinogens.

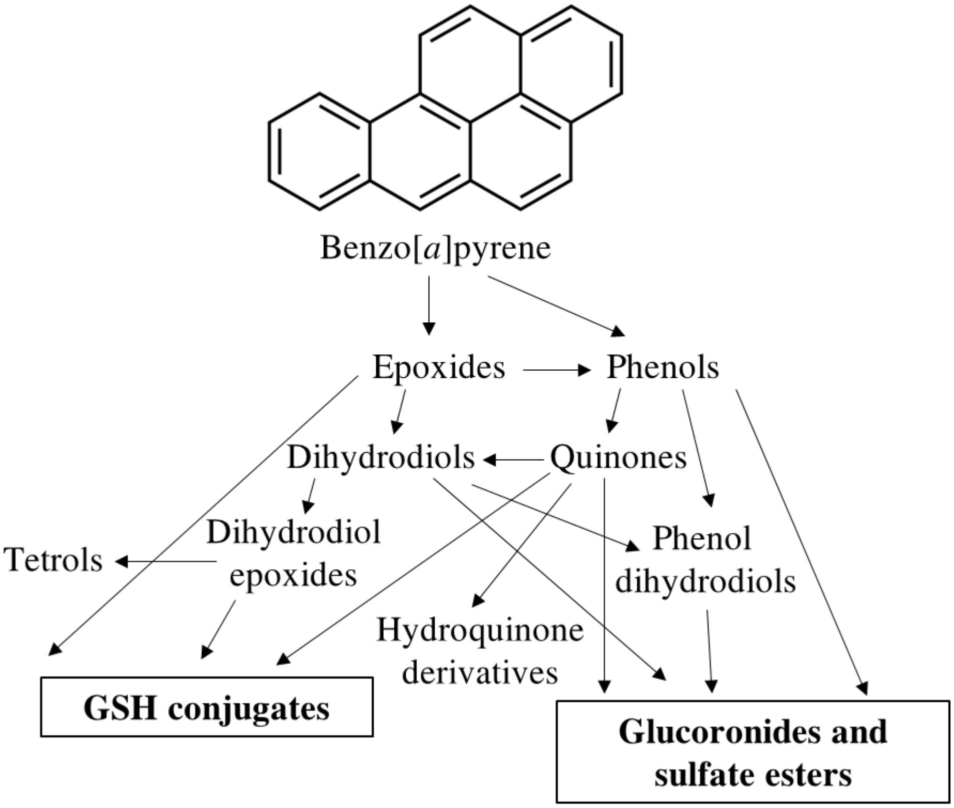

Figure 1-3 Metabolic pathway scheme for benzo[*a*]pyrene. Adapted from IARC monograph 92 (IARC 2010) and Hecht (1999). GSH is glutathione.

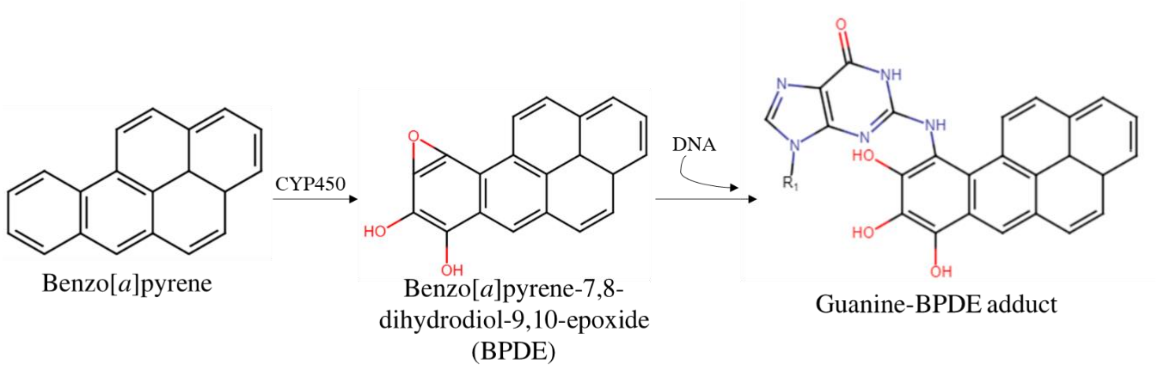

Figure 1-4 A benzo[*a*]pyrene metabolism pathway leading to a guanine adduct. Adapted from Perera et al. (2015).

There are three main routes of formation of DNA damaging PAH metabolites: diol-epoxide, radical cation, and quinone-semi-quinones. Diol-epoxides are thought of as the most prominent pathway of production of DNA damage, with the diol-epoxide derivative of BaP (i.e.,BPDE) being most harmful metabolite (IARC 2010). DNA adducts of other PAHs have also been identified; however, they are less efficiently produced (Hecht et al. 2010; Jeffrey et al. 1977; Kim et al. 1998; Lodovici et al. 1998). The diol-epoxide pathway is more commonly used in PAHs with sterically hindered regions (i.e., three or four angularly fused benzo rings creating a sterically-hindered region called a bay or fjord region, respectively) such as in BaP or dibenzo[*a,l*]pyrene. Due to steric hinderance, the electrophilic dihydrodiol epoxide intermediates, which are formed from PAHs with bay or fjord regions, are highly reactive and relatively resistant to phase II conjugation that contributes to elimination from the body, (IARC 2010; Jerina et al. 2012). Radical cations can also be formed when P450 enzymes remove a π electron from a double bond in a PAH. The third route generates PAH-ortho-quinones, formed from the dehydrogenation of PAH dihydrodiols by aldo-keto reductases (AKRs) (Benigni 2003). The electrophilic compounds formed during PAH metabolism are DNA-reactive, resulting in the formation of bulky adducts. PAH-DNA adducts can often be removed via nucleotide excision repair, but unsuccessful repair or errors during DNA synthesis can ultimately lead to mutations (Braithwaite, Wu, and Wang 1999; Ewa and Danuta 2017). PAH-induced mutations can initiate important tumor formation events including activation of proto-oncogenes or inactivation of tumor suppressor genes (Melendez-Colon et al. 1999). These mechanisms are thought to contribute to the carcinogenicity of

PAHs such as BaP, dibenzo[*a,l*]pyrene, 7,12-dimethylbenz[*a*]-anthracene and 3-methylcholanthrene (IARC 2010).

DNA adducts are not the only way PAHs can cause DNA damage. Reactive oxygen species (ROS) can also induce DNA damage via oxidative stress. For example, BaP derivatives can enter redox cycles, with ortho-quinones generating ROS that contributes to the oxidation of purines (Baulig et al. 2003; Briedé et al. 2009; Park et al. 2005). Oxidative DNA damage can contribute to carcinogenicity via modulation of gene expression and induction of mutations (Valavanidis et al. 2013). Another PAH-related pathophysiological pathway involves activation of estrogen receptors (ER). Several PAHs have been shown to be inducers of ER-mediated activity, and both BaP and benz[*a*]anthracene have been shown to stimulate cell proliferation through ER activation (Plíšková et al. 2005; Vondráček, Kozubik, and Machala 2002).

Genetic polymorphisms have been investigated for their involvement in conferring susceptibility or resistance to the genotoxic effects of PAHs. Cytochrome P450 1A1 (CYP1A1) is an important enzyme in PAH metabolism; several alleles have been identified for the CYP1A1 gene, resulting in differences in frequency and expression, and ultimately, cancer risk. For example, allele frequencies of *CYP1A1*2A* and *2B have been reported as 6.6% and 2.2% in a Polish population, 18.1% and 8.9% in a Turkish population, and 33% and 19.8% in Japanese (Aynacioglu et al. 1998; Mrozikiewicz et al. 1997; Nakachi et al. 1991). Relatedly, epidemiological data have shown cancer susceptibilities for certain allele carriers, e.g., for lung cancer for carriers of *CYP1A1*2A* and **2B* alleles, and endometrial cancer for carriers of the *CYP1A1*4* allele (Esteller et al. 1997; Kawajiri et al. 1993; Nakachi et al. 1991). Polymorphisms of the

Phase II detoxification enzyme sulfotransferase 1A1 have also been associated with increased breast cancer risk (Tang et al. 2003). In addition to metabolism enzymes, polymorphisms in DNA repair genes (i.e., nucleotide excision repair) have also been shown in increase cancer risk associated with PAH exposure (Binkova et al. 2007; Terry et al. 2004).

In addition to mutagenicity and carcinogenicity, PAHs can elicit other toxicological effects. PAHs have also been shown to be teratogenic, immunotoxic, reprotoxic, neurotoxic, and to cause endocrine-disrupting effects (Sun et al. 2021). For example, a recent review by Lee and Choi (2022) reported PAHs to have been associated with lower IQ, impaired cognitive development, decreased pulmonary function, hypertension, preterm birth, and delayed fetal growth. Other health effects, including respiratory diseases (e.g., bronchitis, allergic reactions), cardiovascular disease (e.g., myocardial infarction, ischemic heart disease, atherosclerosis, peripheral arterial disease, heart rate variation, cardiac autonomic dysfunction), obesity, and infertility have also been associated with PAH exposure. However, there is limited information about these effects, warranting further investigation (Lee and Choi 2022).

Biomonitoring of PAH exposures

There are several ways to assess an individual's PAH exposure. Human biomonitoring of PAHs can involve the measurement of the parent PAH, measurement of PAH-derived DNA or protein adduct, or measurement of a PAH metabolite; all have been measured in a variety of sample types. Unmetabolized PAHs have been measured in saliva, exhaled breath, peripheral blood, umbilical cord blood, human placentas, breast milk, hair, and urine (Fent et al. 2014; Lin et al. 2020; Madhavan and Naidu 2016; Martín

Santos et al. 2020; Motorykin et al. 2015; Pulkrabova et al. 2016; Sobus et al. 2009). PAH adducts have been measured in blood, (e.g., hemoglobin and albumin from peripheral blood and umbilical cord blood) (Bocskay et al. 2005; Kang et al. 1995; Scherer et al. 2000), urothelial cells (Peters et al. 2008; Vineis et al. 1996), and tissue samples such as breast (Tang et al. 2003), esophagus, prostate, cervix, vulva, and placenta (Pratt et al. 2011). Most metabolised PAHs end up in excrement, thus measurement of urinary metabolites are most often used for PAH biomonitoring; it is a convenient, non-invasive biomonitoring approach. Indeed, urinary PAH metabolite monitoring has been used to assess exposures resulting from a variety of activities, including firefighting and building construction (Fent et al. 2020; Hoppe-Jones et al. 2021; Sobus et al. 2009; Wingfors et al. 2018); levels are affected by numerous demographic and lifestyle factors such as smoking status, age of home, and air pollution (Keir et al. 2020; Levine et al. 2015; Strickland and Kang 1999; Wei et al. 2016). However, it should be noted that urinary metabolites are primarily derived from low MW PAHs; the higher MW PAHs, which are often the more carcinogenic and mutagenic, are primarily excreted via feces. For example, ^{14}C-BaP orally administered in lactating goats resulted in 88.2% of the compound in the feces and only 6.3% in urine (Grova et al. 2002). Human fecal sample collection can be socially awkward for both participant and researcher, thus urinary sampling is most commonly used for PAH biomonitoring.

Silicone Wristbands

O’Connell et al. (2014) described the use of silicone wristbands (SWBs) as personal, passive samplers that efficiently and conveniently assess exposures to a variety of toxicants (e.g., PAHs, pesticides). SWs can be used to collect environmental organic molecules through passive diffusion, and provide time-weighted averages of environmental chemical concentrations. Studies have employed SWs to monitor exposures to numerous substances, such as pesticides in farmers (Donald et al. 2016), polybrominated diphenyl ethers (PBDEs) and organophosphorus flame retardants (OPFRs) in children (Hammel et al. 2020; Kile et al. 2016), and PAHs, PBDEs, brominated FRs, and OPFRs in adults (Wang et al. 2019). SWs, which are used for air monitoring, are thought to provide information on respiratory exposures. However, they also provide information on dermal and dietary exposures from surface deposition and dissolution in sweat (Hamzai et al. 2021). SWBs offer a simple, convenient way to assess individuals' PAH exposures.

To date, several studies have used silicone passive sampling devices for occupational monitoring of airborne toxicants present at the scene of municipal structural fires. Baum et al. (2020) and Caban-Martinez et al. (2020) employed the SW approach to show that firefighters are exposed to PAHs. Bakali et al. (2021) used stationary passive silicone samplers to assess the distribution of PAHs emitted by live fire training with different fuels. Using a modified approach, Poutasse et al., (2022, 2020) used military style "dog-tags" to show that firefighters are exposed to PAHs and endocrine disrupting chemicals. Most recently, Levasseur et al. (2022) measured 134 compounds, including PAHs, OPFRs, PBDEs, and PFAS, in SW worn by firefighters while on-shift. Baum et al., (2020) also used SW to measure off-gassing PAHs from firefighter bunker gear by placing the gear and SW in an airtight case for 24h; measurable PAHs were found to off-gas with significantly more off-gassing after a recent fire exposure. To date the SWB approach has not been used to survey toxicants, including PAHs, specifically encountered during live firefighting activities nor with matching air and biological samples.

Firefighters' exposures to combustion emissions

Monitoring firefighters' exposures to chemicals of concerns is essential for quantifying exposure levels; moreover, for planning appropriate follow up studies, and ultimately, determining courses of action to effectively reduce exposure.

Firefighters' PAH exposures

Firefighters' occupational PAH exposures have been measured via dermal surface sampling and/or sampling of bodily fluids such as blood and urine. Using active air samplers, passive SW and silicone dog tags, elevated environmental concentrations of PAHs have been measured at fire suppression events, inside fire trucks, in fire station air and settled dust (Banks et al. 2020; Caban-Martinez et al. 2020; Keir et al. 2020; Oliveira et al. 2017; Poutasse et al. 2020). For example, PAH levels in air have been observed to be as high as 22,000 µg m^{-3} at controlled structural burns and 28,600 µg m^{-3} at emergency structural fires (Fent et al. 2014; Keir et al. 2020). With respect to surfaces, several studies have found significantly higher PAH levels on firefighters' skin and personal protective equipment (PPE) after a fire suppression event as compared to before (Fent et al. 2014, 2017; Keir et al. 2020; Wingfors et al. 2018). Internally, urinary PAH metabolite concentrations are most often used to assess internal dose since the sample collection procedure is non-invasive and more convenient compared to blood, biopsy sampling, etc. Many studies have found significantly elevated urinary PAH metabolites concentrations for firefighters at controlled/training fires, emergency fires, and wildland fires, using paired pre- and post-fire samples (Ekpe et al. 2021; Fent et al. 2017, 2020;

Hoppe-Jones et al. 2021; Keir, et al. 2017; Oliveira et al. 2016; Wingfors et al. 2018). Interestingly, Keir et al. (2017) found that even before a fire, firefighters had elevated concentrations of some urinary PAH metabolites compared to the general population. Firefighters’ exposures to PAHs are evident, but the best courses of action to reduce such exposures are not as clear.

Since use of a self-contained breathing apparatus (SCBA) prevents respiratory exposure when worn properly (i.e., protection factor of 10,000), the main route of firefighters’ PAH exposure is thought to be via dermal contact (Bollinger and Schutz 1987). Indeed, previous work has found significant increases in PAHs on skin after firefighting, and relationships between dermal contamination and urinary PAH metabolites (Fent et al. 2014, 2017; Fernando et al. 2016; Keir, et al. 2017; Wingfors et al. 2018). Interestingly, dermal exposure to combustion by-products is thought to be involved in first reported case of occupational cancer by Percivall Pott in 1775; that study empirically connected scrotum cancer and soot exposure in young chimney sweeps (Pott 1775). More contemporary investigations have shown that many of the PAHs classified by IARC as probably or possibly carcinogenic to humans (i.e., IARC Group 2A or 2B) have been shown to cause skin cancer and/or be initiators of skin cancer in rodents (IARC 1983, 2010).

Given the weight of evidence above supporting the notion that firefighters are dermally exposed to PAHs, the use of skin cleaning wipes after fire suppression activities has become more prevalent, in an effort to reduce overall exposure and internal dose. The intention is to reduce post-exposure dermal absorption of carcinogens by removal of contaminants deposited on skin as soon as possible, rather than waiting to shower upon return to the fire station. However, only one study to date has investigated the efficacy of dermal cleaning procedures for reduction of PAH exposures. Fent et al. (2017) found that baby wipes were able to significantly reduce post-exposure PAH levels on firefighters' necks by a median of 54%. That being said, the authors only considered external PAH contamination levels and did not consider PAH absorption and internal dose (e.g., urinary metabolite concentrations). Moreover, skin-cleaning wipes can contain a range of ingredients, and it is not clear whether the specific types of wipes and/or wiping procedures can alter the ability to remove PAHs deposited on skin.

Despite the aforementioned importance of dermal contact, it is evident that firefighters are exposed to PAHs via multiple routes. Although the self-contained breathing apparatus (SCBA) that firefighters wear protects them from respiratory exposure to contaminated air, SCBA are heavy and cumbersome thus aren't always worn; they are often removed as soon as knockdown (i.e., extinction of flames) is complete. Thus, firefighters' respiratory exposure to substances such as PAHs cannot be ignored. In fact, Andersen et al. (2017) found that firefighters' highest exposures to particulate matter was during situations where they removed their SCBA while in "safe zones". Furthermore, Fent et al. (2015) found a significant relationship between the levels of organic compounds in firefighters' post-burn exhaled breath and airborne concentrations of compounds off-gassing from firefighters' used PPE (K. Fent et al., 2015).

Summary

This thesis aimed to assess firefighters' exposures to PAHs; moreover, ways to reduce exposures to harmful combustion products via dermal decontamination. A multifaceted approach was taken, using several methodologies to assess PAH exposures using active air sampling, skin and PPE surface sampling at different time points, and urinary biomonitoring for metabolites and mutagenic potency. The thesis is written as manuscripts and divided into three data chapters. Chapter two contains the results of a study that investigated surface and air contamination with PAHs and metals at fire stations and emergency fire events. Chapter three is comprised of a study that uses silicone wristbands as passive samplers to assess PAH exposure of firefighters at live fire training. Chapter four presents an intervention study that assesses the effectiveness of post-fire dermal surface decontamination to reduce the internal dose of PAHs after live fire training. Chapter five provides concluding remarks.

Two cohorts of firefighting participants were recruited, and details of the recruitment process can be found in their respective chapters. Data for Chapter 2 was collected as part of a companion study published by Keir et al. (2017). Chapters 3 and 4 involve the same cohort; data from the silicone wristbands worn by the firefighters are presented in Chapter 3, and the results from the dermal decontamination interventions are presented in Chapter 4.

References

Alalaiwe, Ahmed, Yin Ku Lin, Chih Hung Lin, Pei Wen Wang, Jie Yu Lin, and Jia You Fang. 2020. “The Absorption of Polycyclic Aromatic Hydrocarbons into the Skin to Elicit Cutaneous Inflammation: The Establishment of Structure–Permeation and in Silico–in Vitro–in Vivo Relationships.” *Chemosphere* 255:126955.

Andersen, Maria Helena Guerra, Anne Thoustrup Saber, Peter Bøgh Pedersen, Steffen Loft, Åse Marie Hansen, Ismo Kalevi Koponen, Julie Elbæk Pedersen, Niels Ebbehøj, Eva Carina Nørskov, Per Axel Clausen, Anne Helene Garde, Ulla Vogel, and Peter Møller. 2017. “Cardiovascular Health Effects Following Exposure of Human Volunteers during Fire Extinction Exercises.” *Environmental Health: A Global Access Science Source* 16(1):1–9.

Armstrong, Ben, Emma Hutchinson, John Unwin, and Tony Fletcher. 2004. “Lung Cancer Risk after Exposure to Polycyclic Aromatic Hydrocarbons: A Review and Meta-Analysis.” *Environmental Health Perspectives* 112(9):970–78.

Austin, C. C., D. Wang, D. J. Ecobichon, and G. Dussault. 2001. “Characterization of Volatile Organic Compounds in Smoke at Municipal Structural Fires.” *Journal of Toxicology and Environmental Health, Part A* 63(6):437–58.

Aynacioglu, A. Sükrü, Ingolf Cascorbi, Przemyslaw M. Mrozikiewicz, and Ivar Roots. 1998. “High Frequency of CYP1A1 Mutations in a Turkish Population.” *Archives of Toxicology 1998 72:4* 72(4):215–18.

Bakali, Umer, Jeramy L. R. Baum, Chitvan Killawala, Erin N. Kobetz, Natasha Schaefer Solle, Sapna K. Deo, Alberto J. Caban-Martinez, Leonidas G. Bachas, and Sylvia Daunert. 2021. “Mapping Carcinogen Exposure across Urban Fire Incident Response Arenas Using Passive Silicone-Based Samplers.” *Ecotoxicology and Environmental Safety* 228:112929.

Banks, Andrew P. W., Michelle Engelsman, Chang He, Xianyu Wang, and Jochen F. Mueller. 2020. “The Occurrence of PAHs and Flame-Retardants in Air and Dust from Australian Fire Stations.” *Occupational and Environmental Hygiene* 17(2–3):73–84.

Banks, Andrew P. W., Phong Thai, Michelle Engelsman, Xianyu Wang, Andres F. Osorio, and Jochen F. Mueller. 2021. “Characterising the Exposure of Australian Firefighters to Polycyclic Aromatic Hydrocarbons Generated in Simulated Compartment Fires.” *International Journal of Hygiene and Environmental Health* 231:113637.

Bartsch, N., J. Heidler, B. Vieth, C. Hutzler, and A. Luch. 2016. “Skin Permeation of Polycyclic Aromatic Hydrocarbons: A Solvent-Based in Vitro Approach to Assess Dermal Exposures against Benzo[a]Pyrene and Dibenzopyrenes.” *Journal of Occupational and Environmental Hygiene* 13(12):969–79.

Baulig, Augustin, Michèle Garlatti, Véronique Bonvallot, Alexandre Marchand, Robert Barouki, Francelyne Marano, and Armelle Baeza-Squiban. 2003. “Involvement of Reactive Oxygen Species in the Metabolic Pathways Triggered by Diesel Exhaust Particles in Human Airway Epithelial Cells.” *American Journal of Physiology - Lung Cellular and Molecular Physiology* 285(3 29-3):671–79.

Baum, Jeramy L. R., Umer Bakali, Chitvan Killawala, Katerina M. Santiago, Emre Dikici, Erin N. Kobetz, Natasha Schaefer Solle, Sapna Deo, Leonidas Bachas, and

Sylvia Daunert. 2020. "Evaluation of Silicone-Based Wristbands as Passive Sampling Systems Using PAHs as an Exposure Proxy for Carcinogen Monitoring in Firefighters: Evidence from the Firefighter Cancer Initiative." *Ecotoxicology and Environmental Safety* 205:111100.

Benigni, Romualdo. 2003. *Quantitative Structure-Activity Relationship (QSAR) Models of Mutagens and Carcinogens*. Boca Raton, FL: CRC press.

Bevan, D. R. and M. R. Ulman. 1991. "Examination of Factors That May Influence Disposition of Benzo[a]Pyrene in Vivo: Vehicles and Asbestos." *Cancer Letters* 57(2):173–79.

Binkova, Blanka, Irena Chvatalova, Zdena Lnenickova, Alena Milcova, Elena Tulupova, Peter B. Farmer, and Radim J. Sram. 2007. "PAH–DNA Adducts in Environmentally Exposed Population in Relation to Metabolic and DNA Repair Gene Polymorphisms." *Mutation Research/Fundamental and Molecular Mechanisms of Mutagenesis* 620(1–2):49–61.

Bocskay, Kirsti A., Deliang Tang, Manuela A. Orjuela, Xinhua Liu, Dorothy P. Warburton, and Frederica P. Perera. 2005. "Chromosomal Aberrations in Cord Blood Are Associated with Prenatal Exposure to Carcinogenic Polycyclic Aromatic Hydrocarbons." *Cancer Epidemiology, Biomarkers & Prevention* 14(2):506–11.

Bollinger, N. J. and R. H. Schutz. 1987. *NIOSH Guide to Industrial Respiratory Protection*. Cincinnati, OH.

Bourgart, Etienne, Renaud Persoons, Marie Marques, Alex Rivier, Franck Balducci, Anne von Koschembahr, David Béal, Marie Thérèse Leccia, Thierry Douki, and Anne Maitre. 2019. "Influence of Exposure Dose, Complex Mixture, and Ultraviolet Radiation on Skin Absorption and Bioactivation of Polycyclic Aromatic Hydrocarbons Ex Vivo." *Archives of Toxicology* 93(8):2165–84.

Braithwaite, Elena, Xiaohua Wu, and Zhigang Wang. 1999. "Repair of DNA Lesions: Mechanisms and Relative Repair Efficiencies." *Mutation Research/Fundamental and Molecular Mechanisms of Mutagenesis* 424(1–2):207–19.

Briedé, Jacob J., Roger W. L. Godschalk, Marijn T. G. Emans, Theo M. C. M. De Kok, Ebienus Van Agen, Jan M. S. Van Maanen, Frederik Jan Van Schooten, and Jos C. S. Kleinjans. 2009. "In Vitro and In Vivo Studies on Oxygen Free Radical and DNA Adduct Formation in Rat Lung and Liver during Benzo[a]Pyrene Metabolism." *Https://Doi.Org/10.1080/10715760400000976* 38(9):995–1002.

Brinkmann, Joep, Kristin Stolpmann, Susanne Trappe, Timo Otter, Doris Genkinger, Udo Bock, Manfred Liebsch, Frank Henkler, Christoph Hutzler, and Andreas Luch. 2013. "Metabolically Competent Human Skin Models: Activation and Genotoxicity of Benzo[a]Pyrene." *Toxicological Sciences* 131(2):351–59.

Caban-Martinez, Alberto J., Paola Louzado-Feliciano, Katerina M. Santiago, Jeramy Baum, Natasha Schaefer Solle, Geovanny Rivera, Marija Miric, Eddy Perez-Then, Erin N. Kobetz-Kerman, and Sylvia Daunert. 2020. "Objective Measurement of Carcinogens Among Dominican Republic Firefighters Using Silicone-Based Wristbands." *Journal of Occupational and Environmental Medicine* 62(11):e611–15.

Centers for Disease Control and Prevention. 2006. "Fatalities among Volunteer and Career Firefighters--United States, 1994-2004." *MMWR Morb. Mortal. Wkly. Rep.* 55(16):453–55.

Collin, M. Samuel, Senthil Kumar Venkatraman, Naveensubramaniam Vijayakumar, V. Kanimozhi, S. Muhammad Arbaaz, R. G. Sibiya Stacey, Jogannagari Anusha, Rajan Choudhary, Vladislav Lvov, Gabriel Ibrahin Tovar, Fedor Senatov, Sivasankar Koppala, and Sasikumar Swamiappan. 2022. "Bioaccumulation of Lead (Pb) and Its Effects on Human: A Review." Journal of Hazardous Materials Advances 7:100094.

Daniels, Robert D., Travis L. Kubale, James H. Yiin, Matthew M. Dahm, Thomas R. Hales, Dalsu Baris, Shelia H. Zahm, James J. Beaumont, Kathleen M. Waters, and Lynne E. Pinkerton. 2014. "Mortality and Cancer Incidence in a Pooled Cohort of US Firefighters from San Francisco, Chicago and Philadelphia (1950–2009)." *Occupational and Environmental Medicine* 71(6):388–97.

Demers, Paul A., David M. DeMarini, Kenneth W. Fent, Deborah C. Glass, Johnni Hansen, Olorunfemi Adetona, Maria HG Andersen, Laura E. Beane Freeman, Alberto J. Caban-Martinez, Robert D. Daniels, Timothy R. Driscoll, Jaclyn M. Goodrich, Judith M. Graber, Tracy L. Kirkham, Kristina Kjaerheim, David Kriebel, Alexandra S. Long, Luana C. Main, Marta Oliveira, Susan Peters, Lauren R. Teras, Emily R. Watkins, Jefferey L. Burgess, Anna A. Stec, Paul A. White, Nathan L. DeBono, Lamia Benbrahim-Tallaa, Aline de Conti, Fatiha El Ghissassi, Yann Grosse, Leslie T. Stayner, Eero Suonio, Susana Viegas, Roland Wedekind, Pauline Boucheron, Bayan Hosseini, Joanne Kim, Hana Zahed, Heidi Mattock, Federica Madia, and Mary K. Schubauer-Berigan. 2022. "Carcinogenicity of Occupational Exposure as a Firefighter." *The Lancet Oncology* 23(8):985–86.

Donald, Carey E., Richard P. Scott, Kathy L. Blaustein, Mary L. Halbleib, Makhfousse Sarr, Paul C. Jepson, and Kim A. Anderson. 2016. "Silicone Wristbands Detect Individuals' Pesticide Exposures in West Africa." *Royal Society Open Science* 3(8).

Ekpe, Okon Dominic, Wonjin Sim, Sol Choi, Gyojin Choo, and Jeong Eun Oh. 2021. "Assessment of Exposure of Korean Firefighters to Polybrominated Diphenyl Ethers and Polycyclic Aromatic Hydrocarbons via Their Measurement in Serum and Polycyclic Aromatic Hydrocarbon Metabolites in Urine." *Environmental Science and Technology* 55(20):14015–25.

Esteller, Manel, Angel Garcia, Josep Maria Martinez-Palones, Jordi Xercavins, and Jaume Reventos. 1997. "Germ Line Polymorphisms in Cytochrome-P450 1A1 (C4887 CYP1A1) and Methylenetetrahydrofolate Reductase (MTHFR) Genes and Endometrial Cancer Susceptibility." *Carcinogenesis* 18(12):2307–11.

Ewa, Błaszczyk and Mielżyńska Švach Danuta. 2017. "Polycyclic Aromatic Hydrocarbons and PAH-Related DNA Adducts." *Journal of Applied Genetics* 58(3):321–30.

Fabian, Thomas Z., Jacob L. Borgerson, Pravinray D. Gandhi, C. Stuart Baxter, Clara Sue Ross, James E. Lockey, and James M. Dalton. 2014. "Characterization of Firefighter Smoke Exposure." Fire Technology 50(4):993–1019.

Fent, K. W., B. Alexander, J. Roberts, S. Robertson, C. Toennis, D. Sammons, S. Bertke, S. Kerber, D. Smith, and G. Horn. 2017. "Contamination of Firefighter Personal Protective Equipment and Skin and the Effectiveness of Decontamination Procedures." *Journal of Occupational and Environmental Hygiene* 14(10):801–14.

Fent, Kenneth W., Judith Eisenberg, John Snawder, Deborah Sammons, Joachim D. Pleil, Matthew A. Stiegel, Charles Mueller, Gavin P. Horn, and James Dalton. 2014. "Systemic Exposure to PAHs and Benzene in Firefighters Suppressing Controlled

Structure Fires." *The Annals of Occupational Hygiene* 58(7):830–45.

Fent, Kenneth W., Christine Toennis, Deborah Sammons, Shirley Robertson, Stephen Bertke, Antonia M. Calafat, Joachim D. Pleil, M. Ariel Geer Wallace, Steve Kerber, Denise Smith, and Gavin P. Horn. 2020. "Firefighters' Absorption of PAHs and VOCs during Controlled Residential Fires by Job Assignment and Fire Attack Tactic." *Journal of Exposure Science & Environmental Epidemiology* 30(2):338–49.

Fernando, Sujan, Michael Gallea, Lori VandenEnden, Ron House, Dave Verma, Lorraine Shaw, Brian E. McCarry, Don Shaw, and Philip Britz-McKibbin. 2016. "Evaluation of Firefighter Exposure to Wood Smoke During Training Exercises at Burn Houses." *Environmental Science and Technology* 50(3):1536–43.

Foa, V., G. Elia, N. Schiavulli, P. Sartorelli, G. Schiarra, A. Cenni, M. T. Novelli, F. Mangani, G. Cecchetti, and R. Iachetta. 1998. "[Non-Bioavailability of Carcinogenic Polycyclic Aromatic Hydrocarbons Contained in Coal Dust]." *La Medicina Del Lavoro* 89(1):68–77.

Gerde, P., M. A. Medinsky, and J. A. Bond. 1991. "The Retention of Polycyclic Aromatic Hydrocarbons in the Bronchial Airways and in the Alveolar Region—A Theoretical Comparison." *Toxicology and Applied Pharmacology* 107(2):239–52.

Gerde, P., B. A. Muggenburg, M. Lundborg, and A. R. Dahl. 2001. "The Rapid Alveolar Absorption of Diesel Soot-Adsorbed Benzo[a]Pyrene: Bioavailability, Metabolism and Dosimetry of an Inhaled Particle-Borne Carcinogen." *Carcinogenesis* 22(5):741–49.

Gerde, Per, Bruce A. Muggenburg, and Rogene F. Henderson. 1993. "Disposition of Polycyclic Aromatic Hydrocarbons in the Respiratory Tract of the Beagle Dog: III. Mechanisms of the Dosimetry." *Toxicology and Applied Pharmacology* 121(2):328–34.

Grimmer, G., H. Brune, G. Dettbarn, K. W. Naujack, U. Mohr, and R. Wenzel-Hartung. 1988. "Contribution of Polycyclic Aromatic Compounds to the Carcinogenicity of Sidestream Smoke of Cigarettes Evaluated by Implantation into the Lungs of Rats." *Cancer Letters* 43(3):173–77.

Grova, Nathalie, Cyril Feidt, Claire Laurent, and Guido Rychen. 2002. "[14C] Milk, Urine and Faeces Excretion Kinetics in Lactating Goats after an Oral Administration of [14C]Polycyclic Aromatic Hydrocarbons." *International Dairy Journal* 12(12):1025–31.

Guidotti, Tee L. 2016. *Health Risks and Fair Compensation in the Fire Service. Risk, Systems and Decisions.* Switzerland: Springer International Publishing.

Gulliver, S. B., R. T. Zimering, F. Dobani, M. L. Pennington, S. B. Morissette, B. W. Kamholz, J. A. Knight, T. M. Keane, N. A. Kimbrel, T. P. Carpenter, and E. C. Meyer. 2019. "Alcohol Use and Mental Health Symptoms in Female Firefighter Recruits." *Occupational Medicine* 69(8–9):625–31.

Hadgraft, Jonathan. 2004. "Skin Deep." *European Journal of Pharmaceutics and Biopharmaceutics* 58(2):291–99.

Hammel, Stephanie C., Kate Hoffman, Allison L. Phillips, Jessica L. Levasseur, Amelia M. Lorenzo, Thomas F. Webster, and Heather M. Stapleton. 2020. "Comparing the Use of Silicone Wristbands, Hand Wipes, and Dust to Evaluate Children's Exposure to Flame Retardants and Plasticizers." *Environmental Science and Technology* 54(7):4484–94.

Hamzai, Laila, Nicolas Lopez Galvez, Eunha Hoh, Nathan G. Dodder, Georg E. Matt, and Penelope J. Quintana. 2021. "A Systematic Review of the Use of Silicone Wristbands for Environmental Exposure Assessment, with a Focus on Polycyclic Aromatic Hydrocarbons (PAHs)." *Journal of Exposure Science & Environmental Epidemiology 2021 32:2* 32(2):244–58.

Han, Yong, Yingjun Chen, Yanli Feng, Wenhuai Song, Fang Cao, Yanlin Zhang, Qing Li, Xin Yang, and Jianmin Chen. 2020. "Different Formation Mechanisms of PAH during Wood and Coal Combustion under Different Temperatures." *Atmospheric Environment* 222:117084.

Hansch, C., A. Leo, and D. Hoekman. 1995. *Exploring QSAR - Hydrophobic, Electronic, and Steric Constants*. Washington, DC: American Chemical Society.

Haynes, W. M. 2015. *CRC Handbook of Chemistry and Physics*. 95th ed. Boca Raton, FL: CRC Press LLC.

Hecht, Stephen S. 1999. "Tobacco Smoke Carcinogens and Lung Cancer." *JNCI: Journal of the National Cancer Institute* 91(14):1194–1210.

Hecht, Stephen S., Steven G. Carmella, Peter W. Villalta, and J. Bradley Hochalter. 2010. "Analysis of Phenanthrene and Benzo[a]Pyrene Tetraol Enantiomers in Human Urine: Relevance to the Bay Region Diol Epoxide Hypothesis of Benzo[a]Pyrene Carcinogenesis and to Biomarker Studies." *Chemical Research in Toxicology* 23(5):900–908.

Hoppe-Jones, Christiane, Stephanie C. Griffin, John J. Gulotta, Darin D. Wallentine, Paul K. Moore, Shawn C. Beitel, Leanne M. Flahr, Jing Zhai, Jin J. Zhou, Sally R. Littau, Devi Dearmon-Moore, Alesia M. Jung, Fernanda Garavito, Shane A. Snyder, and Jefferey L. Burgess. 2021. "Evaluation of Fireground Exposures Using Urinary PAH Metabolites." *Journal of Exposure Science & Environmental Epidemiology 2021 31:5* 31(5):913–22.

IARC. 1983. "Polynuclear Aromatic Compounds, Part 1, Chemical, Environmental and Experimental Data." *IARC Monogr. Eval. Carcinog. Risks Hum.* 32(1):1–477.

IARC. 2010. "Some Non-Heterocyclic Polycyclic Aromatic Hydrocarbons and Some Related Exposures." *IARC Monogr. Eval. Carcinog. Risks Hum.* 92:1–853.

Ifegwu, Okechukwu Clinton and Chimezie Anyakora. 2015. "Polycyclic Aromatic Hydrocarbons: Part I. Exposure." *Advances in Clinical Chemistry* 72:277–304.

Jeffrey, A. M., I. B. Weinstein, K. W. Jennette, K. Grzeskowiak, K. Nakanishi, R. G. Harvey, H. Autrup, and C. Harris. 1977. "Structures of Benzo(a)Pyrene–Nucleic Acid Adducts Formed in Human and Bovine Bronchial Explants." *Nature 1977 269:5626* 269(5626):348–50.

Jerina, D. M., H. Yagi, R. E. Lehr, D. R. Thakker, M. Schaefer-Ridder, J. M. Karle, W. Levin, A. W. Wood, R. L. Chang, and A. H. Conney. 2012. "The Bay-Region Theory of Carcinogenesis by Polycyclic Aromatic Hydrocarbons." *Polycyclic Hydrocarbons and Cancer* 1:173–88.

Jitnarin, Nattinee, Walker SC Poston, Christopher K. Haddock, Sara A. Jahnke, and Rena S. Day. 2015. "Tobacco Use Pattern Among a National Firefighter Cohort." *Nicotine & Tobacco Research* 17(1):66–73.

Kang, D. H., N. Rothman, M. C. Poirier, A. Greenberg, C. H. Hsu, B. S. Schwartz, M. E. Baser, J. D. Groopman, A. Weston, and P. T. Strickland. 1995. "Interindividual Differences in the Concentration of 1-Hydroxypyrene-Glucuronide in Urine and

Polycyclic Aromatic Hydrocarbon-DNA Adducts in Peripheral White Blood Cells after Charbroiled Beef Consumption.” *Carcinogenesis* 16(5):1079–85.

Kanitakis, J. 2002. “Anatomy, Histology and Immunohistochemistry of Normal Human Skin.” *European Journal of Dermatology* 12(4):390–401.

Kawajiri, K., K. Nakachi, K. Imai, J. Watanabe, and S. Hayashi. 1993. “The CYP1A1 Gene and Cancer Susceptibility.” *Critical Reviews in Oncology/Hematology* 14(1):77–87.

Kazem, Siamaque, Emma Charlotte Linssen, and Susan Gibbs. 2019. “Skin Metabolism Phase I and Phase II Enzymes in Native and Reconstructed Human Skin: A Short Review.” *Drug Discovery Today* 24(9):1899–1910.

Keir, J. L. A., U. Akhtar, D. M. J. Matschke, T. L. Kirkham, H. M. Chan, P. Ayotte, P. A. White, and J. M. Blais. 2017. “Elevated Exposures to Polycyclic Aromatic Hydrocarbons and Other Organic Mutagens in Ottawa Firefighters Participating in Emergency, On-Shift Fire Suppression.” *Environmental Science and Technology* 51(21):12745–55.

Keir, J. L. A., Umme S. Akhtar, David M. J. Matschke, Tracy L. Kirkham, Hing Man Chan, Pierre Ayotte, Paul A. White, and Jules M. Blais. 2017. “Elevated Exposures to Polycyclic Aromatic Hydrocarbons and Other Organic Mutagens in Ottawa Firefighters Participating in Emergency, On-Shift Fire Suppression.” *Environmental Science & Technology* 51(21):12745–55.

Keir, J. L. A., Umme S. Akhtar, David M. J. Matschke, Paul A. White, Tracy L. Kirkham, Hing Man Chan, and Jules M. Blais. 2020. “Polycyclic Aromatic Hydrocarbon (PAH) and Metal Contamination of Air and Surfaces Exposed to Combustion Emissions during Emergency Fire Suppression: Implications for Firefighters’ Exposures.” *Science of The Total Environment* 698(1):134211.

Keir, JLA, S. Cakmak, JM Blais, and PA White. 2020. “The Influence of Demographic and Lifestyle Factors on Urinary Levels of PAH Metabolites—Empirical Analyses of Cycle 2 (2009–2011) CHMS Data.” *Journal of Exposure Science & Environmental Epidemiology* 31(2):386–97.

Keith, Larry and William Telliard. 1979. “ES&T Special Report: Priority Pollutants: A Perspective View.” *Environmental Science & Technology* 13(4):416–23.

Kile, Molly L., Richard P. Scott, Steven G. O’Connell, Shannon Lipscomb, Megan MacDonald, Megan McClelland, and Kim A. Anderson. 2016. “Using Silicone Wristbands to Evaluate Preschool Children’s Exposure to Flame Retardants.” *Environmental Research* 147:365–72.

Kim, James H., Kevin H. Stansbury, Nigel J. Walker, Michael A. Trush, Paul T. Strickland, and Thomas R. Sutter. 1998. “Metabolism of Benzo[a]Pyrene and Benzo[a]Pyrene-7,8-Diol by Human Cytochrome P450 1B1.” *Carcinogenesis* 19(10):1847–53.

Kirk, Katherine M. and Michael B. Logan. 2015. “Firefighting Instructors’ Exposures to Polycyclic Aromatic Hydrocarbons During Live Fire Training Scenarios.” *Journal of Occupational and Environmental Hygiene* 12(4):227–34.

Kolarsick, Paul A. J., Maria Ann Kolarsick, and Carolyn Goodwin. 2011. “Anatomy and Physiology of the Skin.” *Journal of the Dermatology Nurses’ Association* 3(4):203–13.

Lee, David J., Tulay Koru-Sengul, Monique N. Hernandez, Alberto J. Caban-Martinez, Laura A. McClure, Jill A. Mackinnon, and Erin N. Kobetz. 2020. “Cancer Risk among Career Male and Female Florida Firefighters: Evidence from the Florida Firefighter Cancer Registry (1981-2014).” *American Journal of Industrial Medicine* 63(4):285–99.

Lee, Kyung Joo and Kyungho Choi. 2022. “Non-Carcinogenic Health Outcomes Associated with Polycyclic Aromatic Hydrocarbons (PAHs) Exposure in Humans: An Umbrella Review.” *Exposure and Health* 1–17.

Levasseur, Jessica L., Kate Hoffman, Nicholas J. Herkert, Ellen Cooper, Duncan Hay, and Heather M. Stapleton. 2022. “Characterizing Firefighter’s Exposure to over 130 SVOCs Using Silicone Wristbands: A Pilot Study Comparing on-Duty and off-Duty Exposures.” *Science of The Total Environment* 834:155237.

Levine, Hagai, Tamar Berman, Rebecca Goldsmith, Thomas Göen, Judith Spungen, Lena Novack, Yona Amitai, Tamar Shohat, and Itamar Grotto. 2015. “Urinary Concentrations of Polycyclic Aromatic Hydrocarbons in Israeli Adults: Demographic and Life-Style Predictors.” *International Journal of Hygiene and Environmental Health* 218(1):123–31.

Lim, G., T. Jang, C. Sim, Y. Ahn, and K. Jeong. 2020. “Comparison of Cortisol Level by Shift Cycle in Korean Firefighters.” *International Journal of Environmental Research and Public Health* 17(13):1–14.

Lin, Meiqing, Jian Tang, Shengtao Ma, Yingxin Yu, Guiying Li, Ruifang Fan, Bixian Mai, and Taicheng An. 2020. “Insights into Biomonitoring of Human Exposure to Polycyclic Aromatic Hydrocarbons with Hair Analysis: A Case Study in e-Waste Recycling Area.” *Environment International* 136:105432.

Lodovici, Maura, Victor Akpan, Luca Giovannini, Francesca Migliani, and Piero Dolara. 1998. “Benzo[a]Pyrene Diol-Epoxide DNA Adducts and Levels of Polycyclic Aromatic Hydrocarbons in Autoptic Samples from Human Lungs.” *Chemico-Biological Interactions* 116(3):199–212.

Lönnermark, Anders and Per Blomqvist. 2006. “Emissions from an Automobile Fire.” *Chemosphere* 62(7):1043–56.

Luo, Kesong, Diya Zeng, Yuan Kang, Xunyang Lin, Na Sun, Cheng Li, Mengqi Zhu, Zhenwen Chen, Yu Bon Man, and Hui Li. 2020. “Dermal Bioaccessibility and Absorption of Polycyclic Aromatic Hydrocarbons (PAHs) in Indoor Dust and Its Implication in Risk Assessment.” *Environmental Pollution* 264:114829.

Tylenda, Carolyn A., Francisco A. Tomei Torres, and Dexter W. Sullivan. 2022. “Antimony.” Handbook on the Toxicology of Metals: Fifth Edition 2:23–40.

Ma, Ying Ge, Ying Duan Lei, Hang Xiao, Frank Wania, and Wen Hua Wang. 2010. “Critical Review and Recommended Values for the Physical-Chemical Property Data of 15 Polycyclic Aromatic Hydrocarbons at 25 °C.” *Journal of Chemical and Engineering Data* 55(2):819–25.

Madhavan, N. D. and K. A. Naidu. 2016. “Polycyclic Aromatic Hydrocarbons in Placenta, Maternal Blood, Umbilical Cord Blood and Milk of Indian Women.” *Http://Dx.Doi.Org/10.1177/096032719501400607* 14(6):503–6.

Martín Santos, Patricia, Laura Campo, Luca Olgiati, Elisa Polledri, Miguel del Nogal Sánchez, and Silvia Fustinoni. 2020. “Development of a Method to Profile 2- to 4-Ring Polycyclic Aromatic Hydrocarbons in Saliva Samples from Smokers and Non-Smokers by Headspace-Solid-Phase Microextraction-Gas Chromatography-Triple

Quadrupole Tandem Mass Spectrometry.” *Journal of Chromatography B* 1152:122273.

Melendez-Colon, Victor J., Andreas Luch, Albrecht Seidel, and William M. Baird. 1999. “Cancer Initiation by Polycyclic Aromatic Hydrocarbons Results from Formation of Stable DNA Adducts Rather than Apurinic Sites.” *Carcinogenesis* 20(10):1885–91.

Moorthy, Bhagavatula, Chun Chu, and Danielle J. Carlin. 2015. “Polycyclic Aromatic Hydrocarbons: From Metabolism to Lung Cancer.” *Toxicological Sciences* 145(1):5–15.

Motorykin, Oleksii, Lisandra Santiago-Delgado, Diana Rohlman, Jill E. Schrlau, Barbara Harper, Stuart Harris, Anna Harding, Molly L. Kile, and Staci L. Massey Simonich. 2015. “Metabolism and Excretion Rates of Parent and Hydroxy-PAHs in Urine Collected after Consumption of Traditionally Smoked Salmon for Native American Volunteers.” *Science of The Total Environment* 514:170–77.

Mrozikiewicz, Przemyslaw M., Ingolf Cascorbi, Jürgen Brockmöller, and Ivar Roots. 1997. “CYP1A1 Mutations 4887A, 4889G, 5639C and 6235C in the Polish Population and Their Allelic Linkage, Determined by Peptide Nucleic Acid-Mediated PCR Clamping.” *Pharmacogenetics* 7(4):303–7.

Naeher, L. P., M. Brauer, M. Lipsett, J. T. Zelikoff, C. D. Simpson, J. Q. Koenig, and K. R. Smith. 2007. “Woodsmoke Health Effects: A Review.” *Inhalation Toxicology* 19(1):67–106.

Nakachi, K., K. Imai, S. Hayashi, J. Watanabe, and K. Kawajiri. 1991. “Genetic Susceptibility to Squamous Cell Carcinoma of the Lung in Relation to Cigarette Smoking Dose.” *Cancer Research* 51(19):5177–80.

Nethery, E., Wheeler, A., Fisher, M. et al. 2012. “Urinary polycyclic aromatic hydrocarbons as a biomarker of exposure to PAHs in air: A pilot study among pregnant women” *J Expo Sci Environ Epidemiol* 22, 70–91.

O’Connell, Steven G., Laurel D. Kincl, and Kim A. Anderson. 2014. “Silicone Wristbands as Personal Passive Samplers.” *Environmental Science and Technology* 48(6):3327–35.

Oliveira, Marta, Klara Slezakova, Maria José Alves, Adília Fernandes, João Paulo Teixeira, Cristina Delerue-Matos, Maria do Carmo Pereira, and Simone Morais. 2017. “Polycyclic Aromatic Hydrocarbons at Fire Stations: Firefighters’ Exposure Monitoring and Biomonitoring, and Assessment of the Contribution to Total Internal Dose.” *Journal of Hazardous Materials* 323:184–94.

Oliveira, Marta, Klara Slezakova, Maria José Alves, Adília Fernandes, João Paulo Teixeira, Cristina Delerue-Matos, Maria do Carmo Pereira, and Simone Morais. 2016. “Firefighters’ Exposure Biomonitoring: Impact of Firefighting Activities on Levels of Urinary Monohydroxyl Metabolites.” *International Journal of Hygiene and Environmental Health*.

Park, Jong-Heum, Sridhar Gopishetty, Lawrence M. Szewczuk, Andrea B. Troxel, Ronald G. Harvey, and Trevor M. Penning. 2005. “Formation of 8-Oxo-7, 8-Dihydro-2’-Deoxyguanosine (8-Oxo-DGuo) by PAH o-Quinones: Involvement of Reactive Oxygen Species and Copper (II)/Copper (I) Redox Cycling.” *Chemical Research in Toxicology* 18(6):1026–37.

Pawar, Gopal, Mohamed Abou Elwafa Abdallah, Eugenia Villaverde De Sáa, and Stuart Harrad. 2016. “Dermal Bioaccessibility of Flame Retardants from Indoor Dust and the Influence of Topically Applied Cosmetics.” *Journal of Exposure Science & Environmental Epidemiology 2017 27:1* 27(1):100–105.

Payan, Jean Paul, Michel Lafontaine, Patrice Simon, Fabrice Marquet, Catherine Champmartin-Gendre, Dominique Beydon, and Elisabeth Ferrari. 2008. “In Vivo and in Vitro Percutaneous Absorption of [14C]Pyrene in Sprague Dawley Male Rats: Skin Reservoir Effect and Consequence on Urinary 1-OH Pyrene Excretion.” *Archives of Toxicology* 82(10):739–47.

Perera, Rukshan T., Aaron M. Fleming, Robert P. Johnson, Cynthia J. Burrows, and Henry S. White. 2015. “Detection of Benzo[a]Pyrene-Guanine Adducts in Single-Stranded DNA Using the α-Hemolysin Nanopore.” *Nanotechnology* 26(7):074002.

Peters, S., G. Talaska, B. A. Jonsson, H. Kromhout, and R. Vermeulen. 2008. “Polycyclic Aromatic Hydrocarbon Exposure, Urinary Mutagenicity, and DNA Adducts in Rubber Manufacturing Workers.” *Cancer Epidemiology and Prevention Biomarkers* 17(6):1452–59.

Plíšková, Martina, Jan Vondráček, Bořivoj Vojtěšek, Alois Kozubík, and Miroslav Machala. 2005. “Deregulation of Cell Proliferation by Polycyclic Aromatic Hydrocarbons in Human Breast Carcinoma MCF-7 Cells Reflects Both Genotoxic and Nongenotoxic Events.” *Toxicological Sciences* 83(2):246–56.

Pott, Percivall. 1775. *Chirurgical Observations Relative to the Cataract, the Polypus of the Nose, the Cancer of the Scrotum, the Different Kinds of Ruptures, and the Mortification of the Toes and Feet*. Printed, by TJ Carnegy, for L. Hawes, W. Clarke, and R. Collins, in Pater-noster row.

Poutasse, Carolyn M., Christopher K. Haddock, Walker S. C. Poston, Sara A. Jahnke, Lane G. Tidwell, Emily M. Bonner, Peter D. Hoffman, and Kim A. Anderson. 2022. “Firefighter Exposures to Potential Endocrine Disrupting Chemicals Measured by Military-Style Silicone Dog Tags.” *Environment International* 158:106914.

Poutasse, Carolyn M., Walker S. C. Poston, Sara A. Jahnke, Christopher K. Haddock, Lane G. Tidwell, Peter D. Hoffman, and Kim A. Anderson. 2020. “Discovery of Firefighter Chemical Exposures Using Military-Style Silicone Dog Tags.” *Environment International* 142:105818.

Pratt, M. Margaret, Kaarthik John, Allan B. Maclean, Senait Afework, David H. Phillips, and Miriam C. Poirier. 2011. “Polycyclic Aromatic Hydrocarbon (PAH) Exposure and DNA Adduct Semi-Quantitation in Archived Human Tissues.” *International Journal of Environmental Research and Public Health 2011, Vol. 8, Pages 2675-2691* 8(7):2675–91.

Pulkrabova, Jana, Michal Stupak, Andrea Svarcova, Pavel Rossner, Andrea Rossnerova, Antonin Ambroz, Radim Sram, and Jana Hajslova. 2016. “Relationship between Atmospheric Pollution in the Residential Area and Concentrations of Polycyclic Aromatic Hydrocarbons (PAHs) in Human Breast Milk.” *Science of The Total Environment* 562:640–47.

Ramesh, A., S. A. Walker, D. B. Hood, M. D. Guillen, K. Schneider, and E. H. Weyand. 2004. “Bioavailability and Risk Assessment of Orally Ingested Polycyclic Aromatic Hydrocarbons.” *International Journal of Toxicology* 23(5):301–33.

Reinberg, Alain E., Michael H. Smolensky, Marc Riedel, Cedric Riedel, Eric Brousse, and Yvan Touitou. 2017. “Do Night and Around-the-Clock Firefighters’ Shift Schedules Induce Deviation in Tau from 24 Hours of Systolic and Diastolic Blood

Pressure Circadian Rhythms?” *Chronobiology International* 34(8):1158–74.

Roy, T. A., A. J. Krueger, C. R. Mackerer, W. Neil, A. M. Arroyo, and J. J. Yang. 2006. “SAR Models for Estimating the Percutaneous Absorption of Polynuclear Aromatic Hydrocarbons.” *Http://Dx.Doi.Org/10.1080/10629369808039155* 9(3–4):171–85.

Sartorelli, P., A. Cenni, G. Matteucci, L. Montomoli, M. T. Novelli, and S. Palmi. 1999. “Dermal Exposure Assessment of Polycyclic Aromatic Hydrocarbons: In Vitro Percutaneous Penetration from Lubricating Oil.” *International Archives of Occupational and Environmental Health* 72(8):528–32.

Sartorelli, Pietro, Loretta Montomoli, Antonietta Gerardina Sisinni, Rossana Bussani, Domenico Cavallo, and Vito Foá. 2001. “Dermal Exposure Assessment of Polycyclic Aromatic Hydrocarbons: In Vitro Percutaneous Penetration from Coal Dust.” *Toxicology and Industrial Health* 17(1):17–21.

Scherer, G., S. Frank, K. Riedel, I. Meger-Kossien-…. Biomarkers &. Prevention, and undefined 2000. 2000. “Biomonitoring of Exposure to Polycyclic Aromatic Hydrocarbons of Nonoccupationally Exposed Persons.” *AACR*.

Sobus, J. R., M. D. McClean, R. F. Herrick, S. Waidyanatha, F. Onyemauwa, L. L. Kupper, and S. M. Rappaport. 2009. “Investigation of PAH Biomarkers in the Urine of Workers Exposed to Hot Asphalt.” *The Annals of Occupational Hygiene* 53(6):551–60.

Sritharan, Jeavana, Tracy L. Kirkham, Jill MacLeod, Niki Marjerrison, Ashley Lau, Mamadou Dakouo, Chloë Logar-Henderson, Tenzin Norzin, Nathan L. DeBono, and Paul A. Demers. 2022. “Cancer Risk among Firefighters and Police in the Ontario Workforce.” *Occupational and Environmental Medicine* 79(8):533–39.

Strickland, Paul and Daehee Kang. 1999. “Urinary 1-Hydroxypyrene and Other PAH Metabolites as Biomarkers of Exposure to Environmental PAH in Air Particulate Matter.” *Toxicology Letters* 108(2):191–99.

Strandberg, Bo, Cecilia Osterman, C., Akdevab, H. K., Moldanovad, J., Langerd, S. 2022. “The Use of Polyurethane Foam (PUF) Passive Air Samplers in Exposure Studies to PAHs in Swedish Seafarers”. *Polycyclic Aromatic Compounds*, 42:2, 448-459.

Sun, James D., Ronald K. Wolff, George M. Kanapilly, and Roger O. McClellan. 1984. “Lung Retention and Metabolic Fate of Inhaled Benzo(a)Pyrene Associated with Diesel Exhaust Particles.” *Toxicology and Applied Pharmacology* 73(1):48–59.

Sun, Kailun, Yan Song, Falin He, Mingyang Jing, Jingchun Tang, and Rutao Liu. 2021. “A Review of Human and Animals Exposure to Polycyclic Aromatic Hydrocarbons: Health Risk and Adverse Effects, Photo-Induced Toxicity and Regulating Effect of Microplastics.” *Science of The Total Environment* 773:145403.

Tang, Deliang, Andrew Rundle, Laverne Mooney, Stan Cho, Freya Schnabel, Alison Estabrook, Amalia Kelly, Richard Levine, Hannina Hibshoosh, and Frederica Perera. 2003. “Sulfotransferase 1A1 (SULT1A1) Polymorphism, PAH-DNA Adduct Levels in Breast Tissue and Breast Cancer Risk in a Case-Control Study.” *Breast Cancer Research and Treatment 2003 78:2* 78(2):217–22.

Terry, Mary Beth, Marilie D. Gammon, Fang Fang Zhang, Sybil M. Eng, Sharon K. Sagiv, Andrea B. Paykin, Qiao Wang, Sharon Hayes, Susan L. Teitelbaum, Alfred I. Neugut, and Regina M. Santella. 2004. “Polymorphism in the DNA Repair Gene XPD, Polycyclic Aromatic Hydrocarbon-DNA Adducts, Cigarette Smoking, and

Breast Cancer Risk.” *Cancer Epidemiology, Biomarkers & Prevention* 13(12):2053–58.

Theall, Gail, Magdalena Eisinger, and Dezider Grunberger. 1981. “Metabolism of Benzo[a]Pyrene and DNA Adduct Formation in Cultured Human Epidermal Keratinocytes.” *Carcinogenesis* 2(7):581–87.

Thomas, Larry, Len Garis, and Chris Biantoro. 2020. *Canadian Firefighter Fatality and Injury: Trend Analysis of Association of Workers Compensation Boards of Canada Fatalityand Injury Claims 2006 –2018*.

Toshio Kuroki, T., J. Hosomi, K. Munakata, T. Onizuka, M. Terauchi, and N. Nemoto. 1982. “Metabolism of Benzo(a)Pyrene in Epidermal Keratinocytes and Dermal Fibroblasts of Humans and Mice with Reference to Variation among Species, Individuals, and Cell Types.” *American Association for Cancer Research* 42(5):1859–65.

Valavanidis, Athanasios, Thomais Vlachogianni, Konstantinos Fiotakis, and Spyridon Loridas. 2013. “Pulmonary Oxidative Stress, Inflammation and Cancer: Respirable Particulate Matter, Fibrous Dusts and Ozone as Major Causes of Lung Carcinogenesis through Reactive Oxygen Species Mechanisms.” *International Journal of Environmental Research and Public Health 2013, Vol. 10, Pages 3886-3907* 10(9):3886–3907.

Vanrooij, J. G. M., E. Vinke, J. De Lange, P. L. B. Bruijnzeel, M. M. Bodelier-Bade, J. Noordhoek, and F. J. Jongeneelen. 1995. “Dermal Absorption of Polycyclic Aromatic Hydrocarbons in the Blood-Perfused Pig Ear.” *Journal of Applied Toxicology* 15(3):193–200.

Venus, Matt, Jacqueline Waterman, and Ian McNab. 2010. “Basic Physiology of the Skin.” *Surgery (Oxford)* 28(10):469–72.

Vineis, P., G. Talaska, C. Malaveille, H. Bartsch, T. Martone, P. Sithisarankul, and O. Strickland. 1996. “DNA Adducts in Urothelial Cells: Relationship with Biomarkers of Exposure to Arylamines and Polycyclic Aromatic Hydrocarbons from Tobacco Smoke.” *Int. J. Cancer* 65:314–16.

Vondráček, Jan, Alois Kozubik, and Miroslav Machala. 2002. “Modulation of Estrogen Receptor-Dependent Reporter Construct Activation and G0/G1–S-Phase Transition by Polycyclic Aromatic Hydrocarbons in Human Breast Carcinoma MCF-7 Cells.” *Toxicological Sciences* 70(2):193–201.

De Vos, Annemarie J. B. M., Fabienne Reisen, Angus Cook, Brian Devine, and Philip Weinstein. 2009. “Respiratory Irritants in Australian Bushfire Smoke: Air Toxics Sampling in a Smoke Chamber and during Prescribed Burns.” *Archives of Environmental Contamination and Toxicology* 56(3):380–88.

Wang, M., Chen, Z., Song, W. et al. 2021. « A review on Cadmium Exposure in the Population and Intervention Strategies Against Cadmium Toxicity”. *Bulletin of Environmental Contamination and Toxicology.* 106, 65–74.

Wang, Shaorui, Kevin A. Romanak, William A. Stubbings, Victoria H. Arrandale, Michael Hendryx, Miriam L. Diamond, Amina Salamova, and Marta Venier. 2019. “Silicone Wristbands Integrate Dermal and Inhalation Exposures to Semi-Volatile Organic Compounds (SVOCs).” *Environment International* 132:105104.

Wei, Binnian, K. Udeni Alwis, Zheng Li, Lanqing Wang, Liza Valentin-Blasini, Connie S. Sosnoff, Yang Xia, Kevin P. Conway, and Benjamin C. Blount. 2016. “Urinary

Concentrations of PAH and VOC Metabolites in Marijuana Users." *Environment International* 88:1–8.

Wingfors, Håkan, Jenny Rattfelt Nyholm, Roger Magnusson, and Cecilia Hammar Wijkmark. 2018. "Impact of Fire Suit Ensembles on Firefighter PAH Exposures as Assessed by Skin Deposition and Urinary Biomarkers." *Annals of Work Exposures and Health* 62(2):221–31.

Yamaguchi, Koji, Tetsuya Mitsui, Yoshinori Aso, and Kenji Sugibayashi. 2008. "Structure–Permeability Relationship Analysis of the Permeation Barrier Properties of the Stratum Corneum and Viable Epidermis/Dermis of Rat Skin." *Journal of Pharmaceutical Sciences* 97(10):4391–4403.

Chapter 2 Polycyclic Aromatic Hydrocarbon (PAH) and Metal Contamination of Air and Surfaces Exposed to Combustion Emissions During Emergency Fire Suppression: Implications for Firefighters' Exposures

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Abstract

This study examined occupational exposures of Ottawa firefighters to combustion by-products and selected metals. We measured exposures to polycyclic aromatic hydrocarbons (PAHs), antimony, cadmium, and lead using (1) personal air samplers worn by firefighters during emergency fire suppression; (2) wipe samples from skin, personal clothing, and personal protective equipment (PPE) collected before and after emergency firefighting (n=29), and (3) air samples collected in three fire stations, truck cabs, and one administration office. We assessed OFS PPE decontamination procedures using wipe samples collected before and after laundering (n=12). Air concentrations exceeded occupational exposure limits at two fire events for lead and nine for PAHs. After fire suppression, PAH concentrations were significantly higher on skin and PPE ($p<0.001$), antimony on skin, clothing, and PPE ($p<0.001$, 0.01, and 0.05, respectively), and lead on skin and PPE ($p<0.001$). Air concentrations of PAHs and antimony were significantly higher in vehicle bays compared to the office ($p<0.05$), but significantly lower compared to fire truck cabs ($p<0.05$). Washing PPE was effective in removing, on average, 61% of PAHs, 55% of antimony, 97% of lead, and 90% of cadmium. These results indicate that firefighters are significantly exposed, via multiple routes, to combustion by-products during on-shift fire suppression.

Introduction

Firefighters experience elevated risk of injury, and of developing cancer and other serious diseases (Guidotti, 1993; LeMasters et al., 2006; Pukkala et al., 2014; Tsai et al., 2015). For example, a meta-analysis of 32 studies reported excess cancer risks in firefighters, relative to the general population, of 2.02 (testicular), 1.53 (myeloma), 1.51 (non-Hodgkin's lymphoma), 1.39 (skin) and 1.28 (prostate) (LeMasters et al., 2006). In 2010 the International Agency for Research on Cancer (IARC) conducted a second meta-analysis that prompted the classification of "occupational exposure as a firefighter" as possibly carcinogenic to humans (i.e., Group 2B) (IARC, 2010a). Since the IARC classification, Daniels et al. (2014) investigated cancer rates in a pooled cohort of 29,993 US career firefighters employed between 1950-2009, and found excess cancer mortality (SMR=1.14) and incidence (SIR=1.09) mainly for digestive tract (SMR=1.26, SIR=1.17) and respiratory (SMR=1.10, SIR=1.16) cancers (Daniels et al., 2014). Investigating the causes underlying these empirical results is essential for effective reduction of firefighters' elevated risk of chronic diseases.

Firefighters can be exposed to a variety of carcinogens during firefighting activities. Two groups of chemicals, metals (i.e., antimony, cadmium, and lead) and polycyclic aromatic hydrocarbons (PAHs), are most often studied because of their known toxicities and presence in the air during a fire (Bolstad-Johnson et al., 2000; Lönnermark and Blomqvist, 2006; Guidotti, 2015; Britz-McKibbin et al., 2016). Cadmium has been classified as a Group 1 carcinogen by IARC, and antimony trioxide and lead are classified as possibly carcinogenic to humans (IARC, 1989, 1993, 2006). Several PAHs exhibit carcinogenic activity; benzo[*a*]pyrene is carcinogenic to humans (Group 1), naphthalene, cyclopenta[*c*,*d*]pyrene, dibenz[*a*,*h*]anthracene and dibenzo[*a,l*]pyrene are probably carcinogenic (Group 2A), benz[*j*]aceanthrylene, benz[*a*]anthracene, benzo[*b*]fluoranthene, benzo[*j*]fluoranthene, benzo[*k*]fluoranthene, benzo[*c*]phenanthrene, chrysene, dibenzo[*a,h*]pyrene, dibenzo[*a,i*]-pyrene, indeno[1,2,3-*cd*]pyrene and 5-methylchrysene are possibly carcinogenic (Group 2B) (IARC, 2002, 2010b). PAHs are also known to be mutagenic and teratogenic (IARC, 2010b). In 1976, sixteen PAHs were selected by the US Environmental Protection Agency as priorities for concern and control (Table S2-1) (Keith and Telliard, 1979). With well-defined toxic properties and presence at fires, priority PAHs and selected metals are of utmost concern when it comes to firefighters' chemical exposures during fire suppression.

In general, occupational exposure assessments of municipal firefighters are limited (Caux et al., 2002; Edelman et al., 2003; Baxter et al., 2014). Most previous studies focused on municipal firefighters exposed during training exercises (NIOSH, 2013; Fent et al., 2014; Britz-McKibbin et al., 2016). The relevance of these studies for evaluating exposures during emergency suppression of municipal structural fires is questionable. Emergency fire suppression scenarios are known to be markedly different than training scenarios, particularly in terms of fuel and firefighter behavior. Emergency fires can involve a wide range of fuels (e.g., flooring, upholstery, paints and coatings, bedding, draperies, etc.). Although training fires sometimes include furniture, they are generally restricted with respect to acceptable fuels. In fact, the National Fire Protection Association 1403 Standard for Live Fire Training Evolutions recommends that fuels should only include wood products (National Fire Protection Agency, 2012; Fent et al., 2014). Training scenarios may also differ with respect to PPE use and suppression strategies, since the situations are well-controlled and PPE use is uniform and ubiquitous.

In our earlier study, we employed urinary metabolite analyses to investigate firefighters' exposure to PAHs and other organic mutagens. More specifically, we quantified PAH metabolites and mutagenic activity in urine collected before and after emergency, on-shift fire suppression. The results revealed, on average, a significant 2.9- to 5.3-fold increase in urinary PAH metabolite levels, and a 4.3-fold increase in urinary mutagenicity ($p<0.0001$). Moreover, the results showed that urinary PAH metabolite concentration is empirically related to air and skin PAH contamination. In fact, 54% of the variation in total urinary PAH concentrations can be accounted for by air and skin PAH levels (Keir et al., 2017). This companion paper presents detailed results on the levels and types of PAHs and metals in air and on surfaces (i.e., PPE, clothing, and skin). In addition, we present data regarding the efficacy of PPE decontamination procedures for the removal of PAHs and selected metals.

Materials and Methods

This study was a collaboration between the Ottawa Fire Services (OFS), the University of Ottawa, the University of Toronto, and Health Canada. Before initiation, the study was reviewed and approved by the Research Ethics Boards of the University of Ottawa (i.e., H07-14-01B) and Health Canada (i.e., REB 2014-0035).

Firefighters (n=28) were recruited from four fire stations selected based on their call volume, i.e., to maximize the likelihood of capturing firefighting events. A brochure was circulated to inform potential participants about the study, its rationale, and its objectives. Firefighters interested in participating were enrolled if they met the eligibility criteria: (i) non-smokers, (ii) do not live with smokers, and (iii) agreed to avoid charbroiled (i.e., barbequed) foods and exposures to open fires for the duration of their enrollment. Participants signed a consent form and completed a general questionnaire to assess their work history, health status, and lifestyle. Each volunteer subject participated in one or more study block of five consecutive 24-hour shifts.

Samples from emergency fire responses

Details of sample collection and handling procedures can be found in Keir et al. (2017). Briefly, each firefighter was provided with a sample collection “toolbox” containing all sample collection gear, and an instruction manual outlining the sample collection, handling, and storage procedures. To ensure proper and uniform sample collection and handling, all participants received personalised training from a member of the research team. At the beginning of each shift, participants collected wipe samples, affixed the personal air sampler to their PPE collar, and placed the air sampling pump in the inside left pocket of their PPE coat (Figure S2-1). Polypropylene Tygon tubing running under the PPE coat was used to connect the sampling collection cartridge, containing the glass fiber filter and polyurethane foam (PUF), to the sampling pump. In the event of an emergency fire call, participants removed the cap of the air sampling cartridge and turned on the pump to initiate the collection of personal air. After exiting the scene of the fire, participants stopped the pump, and upon return to their fire station, removed the sample collection cartridge, placed it in a ziplock bag, and stored it in the secure -20°C freezer. All samples were stored in this freezer until collection by university research staff.

The personal air sampler consisted of a GilAir$^{®}$ Plus Personal Air Sampling Pump (Levitt Safety, Ottawa, ON), operated at a 2.5 L/min flow rate, connected to a sampling cartridge consisting of a PUF (URG Corporation, Chapel Hill, NC) and a 25 mm Quartz filter (Whatman, Maidstone, UK) (Figure S2-1). Pumps were calibrated before and after sampling using a Gilian Gilibrator-2 Calibrator (Levitt Safety, Ottawa, ON). All PUFs were cleaned prior to use with 2 static cycles of pressurized fluid extraction at 2000 psi for 5 minutes at 75°C with a 1:1 v/v mixture of Optima grade acetone and hexane; the filters were baked at 400°C for 5 hrs. Respective field blanks were also collected on each day of sample collection. The PUFs and half of the filters were processed for PAHs analyses. The other halves of the filters were processed for metal analyses.

Wipe samples were collected at the beginning of a shift to indicate background contamination, and immediately upon return to the station after the fire suppression event. The following wipe samples were collected: skin (i.e., forehead, neck and wrist), personal clothing worn under PPE (i.e., typically t-shirt and underwear), and PPE (i.e., jacket, pants, belt of self-contained breathing apparatus (SCBA)) (Figure S2-1). Firefighters wore clean, nitrile gloves during sample collection. For PAH analyses, each participant collected wipe samples in the same fashion for PPE, clothing, and skin on their right side using a 30 cm^2 template and AlphaWipes® pre-wetted with 70% isopropyl alcohol (Texwipe Inc., Kernersville, NC, USA).

Left-side samples for metal analyses were collected using Ghost Wipes® moistened with deionized water (SKC Inc., EightyFour, PA, USA). To maximize collection efficiency and consistency, firefighters received detailed wipe sample collection instructions (i.e., all corners of the square template, up and down, side to side and diagonal, and folding before inserting into collection tube). Due to time and logistical constraints, individual wipes were combined by surface type prior to metal and PAH analysis (i.e., forehead neck and wrist for skin, t-shirt and underwear for clothing, and jacket, pants, and SCBA belt for PPE). Firefighters also collected wipe field blanks by exposing a wipe to the ambient environment, and placing it into a labelled sample container. Participants also completed a fire event questionnaire containing questions about the nature of the event, fire intensity and duration of participation in suppression, distance from the fire during suppression, the level of smoke, role in fire suppression, and PPE use.

PPE decontamination samples

This part of the study did not analyze wipes from PPE worn at the studied fire suppression events. Instead, wipe samples were collected from 12 PPE sets used during other fire suppression events in Ottawa (i.e., pre-wash), and then sent to the OFS cleaning facility for decontamination via laundering. Details of the wash cycle can be found in the SI. Matching wipe samples were collected after cleaning (i.e., post-wash) near where the pre-wash samples were collected. Wipe procedures were identical to those used for on-shift PPE wipe sample collection (i.e., 30 cm^2 template with AlphaWipes® and Ghost Wipes®), with the exclusion of a sample from the SCBA belt. Again, samples were pooled (e.g., wipes of PPE jacket and pants were pooled into single pre- and post-washing samples).

Air sampling at fire stations

Air monitoring was conducted at three fire stations (i.e., Fire Stations 1, 2, and 3), and at an OFS administrative office (i.e., reference location). Fire Station 1 is in an urban area next to a busy urban street. Fire Station 2 is near a highway (i.e., < 300 m). Fire Station 3 is in an urban residential area. At each fire station, one sample from the vehicle bay and one within a truck cab were collected for a one hour period on three separate days (i.e., N=3). Three one-hour air samples were also collected at the reference location. We also collected field blanks at each location on each day of sample collection. We collected samples inside the vehicle bays using a modified high-volume air sampler (480 L/min). This sampler contained a polyurethane foam filter (PUF) (Supelco, Pennsylvania, PA) and a Quartz filter (101.6 mm) (Whatman, Maidstone, UK). The personal air sampling devices worn by firefighters (GilAir® Plus Personal Air Sampling Pumps) were also used to collect air samples from fire truck cabs. The PUFs and filters for each sample were cut in half and processed for PAHs and metal analyses separately.

PAH analyses

Detailed information on the PAH analytical method is available from U.S. E.P.A (1994). Briefly, PUFs and filters were extracted using Accelerated Solvent Extraction (ASE 350 and ASE 200, Dionex Corporation, Sunnyvale, CA, USA). All samples were spiked with ^{13}C-labeled standards of the 16 US EPA priority PAHs (Table S2-1) (Cambridge Isotope Laboratories Inc., Andover, MA, USA) prior to extraction. PAHs were separated from interfering compounds by solid-phase extraction (SPE) on SPE cartridges (i.e., 3 mL of alumina (Supelco, Bellefonte, PA) followed by 1 g of silica (Supelco, Bellefonte, PA). To remove water, Alpha wipe extracts underwent liquid-liquid hexane extraction prior to SPE. Extracts were then spiked with an internal standard, p-terphenyl-d14 (Cambridge Isotope Laboratories, Tewksbury, MA 01876), prior to analysis using an HP 6890 gas chromatograph coupled to an HP 5973 mass selective detector (MSD) (Agilent Technologies, Santa Clara, CA, USA). Concentrations of compounds of interest were calculated using isotopic dilution, which accounts for recoveries in each sample by determining PAH-specific recoveries with isotopically labeled standards. Extraction efficiency was determined using linear, equal-weighted standard curves. Analytical method recoveries were determined by spiking the sampling media with a standard mixture of 16 ^{13}C-labeled PAHs (Cambridge Isotope Laboratories Inc., Andover, MA, USA). All samples were method and field blank batch corrected; method detection limit and percent non-detect results are reported in Tables S1 and S2.

Metal analyses

Filters and ghost wipes were digested using a hot nitric acid solution following a modified version of US EPA method 3050B (US EPA, 1996). Samples were dissolved in 10 mL of 1:1 70% HNO_3 and Milli-Q water, and heated on a graphite block (DigiPREP MS block digestion system, SCP Science, QB, Canada) for 30 minutes at 95°C. Once cooled to room temperature, 30% H_2O_2 was added and the samples heated at 95°C for 3 hours. Samples were then diluted to 50 mL with ultra-pure Milli-Q water. All samples were filtered to remove any particulates. In the same manner, a standard reference material (Urban Particulate Matter SRM 1648a from the National Institute of Standards and Technology (NIST, USA)), and blanks (i.e., HNO_3 and Milli-Q H_2O) were prepared to ensure the quality of the analysis. Total metals in the digested extracts were determined using an Agilent ICP-MS 7700 series, with a reactive gas used for removal of known interferences in a complex unknown mixture without loss of sensitivity.

Adjusted occupational exposure limits

Occupational exposure limits (OELs) are usually expressed as a time-weighted average (TWA) based on lifetime exposure during an 8-hour work shift of a 40-hour work-week. Because Ottawa firefighters do not work a typical 8-hour shift, but rather seven to eight 24-hour shifts per 28-day shift cycle, use of the TWA exposure standard may not be appropriate. To compensate for extended work shifts, the Brief and Scala method was applied to calculate adjusted OELs for total PAHs, antimony, cadmium, and lead (i.e., 187.5, 46.8, 9.4, 46.9 $\mu g/m^3$, respectively) (Brief and Scala, 1975; OSHA, 2012; Ontario Ministry of Labour, 2016). Excursion limits (i.e., the concentration to which a worker should not be exposed for more than a total of 30 minutes during a workday, no more than four times per work day, and occurring at least one hour apart) were calculated as 3 times the 8-hour TWA for total PAHs, antimony, cadmium, and lead (i.e., 600, 1500, 30, 150 $\mu g/m^3$, respectively) (ACGIH, 2012; OSHA, 2012; Ontario Ministry of Labour, 2016). TWA values for antimony, cadmium, and lead were obtained from the Ontario Ministry of Labour (Ontario Ministry of Labour, 2016). Due to the lack of PAH OEL values, what is most often used, and what was used herein, is the OEL for coal tar pitch volatiles containing detectable amounts of one or more of benz[*a*]anthracene, benzo[*b*]fluoranthene, chrysene, anthracene, benzo[*a*]pyrene, phenanthrene, acridine, and pyrene (OSHA, 2012).

Data analyses

All samples were field and method blank corrected. If less than 20% of the blank-corrected values for a particular set of samples (e.g., antimony in pre-fire PPE wipes) were below the detection limit, non-detects were assigned a value equal to the detection limit divided by the square root of 2. If more than 20% of the values for a particular sample set were below the detection limit, Robust ROS (regression on order statistics), an approach presented in Helsel (2009), was used to replace non-detect entries. Values were calculated using NDExpo Version 1.0 (http://expostats.ca/site/app-local/NDExpo/) (Helsel, 2009). This method was applied for concentrations of PAHs and metals in personal air, PAHs in fire station air, and cadmium or lead in pre- and post-fire wipe samples of skin and/or PPE. Dibenz[*a,h*]anthracene and benzo[*k*]fluoranthene were not detected in over 80% of air samples collected in OFS offices and fire stations, and were thus omitted from total PAH determinations. The data were log-transformed where appropriate based on visual inspection of box- and Q−Q plots, and results of a Bartlett test for variance homogeneity. Paired (i.e., dependent) t-test was used to evaluate the effect of fire suppression (i.e., pre- versus post-exposure) on surface contamination of skin, clothing, and PPE, and the effectiveness of PPE decontamination. Analysis of variance (ANOVA) with post-hoc multiple range testing (i.e., Duncan method) was employed to examine differences in the mean concentrations of PAHs and metals in air samples from OFS fire stations and reference locations. Data were analyzed using SAS v9.2 for Windows (SAS Institute, Cary, NC, USA). Differences were considered statistically significant when $p < 0.05$.

Results & Discussion

Samples were collected between January and October 2015. Detailed information about the participants is available in Keir et al. (2017). During the study period, 29 air samples with paired wipe samples (i.e., pre- and post-fire) were collected from 16 participants across 18 separate fire suppression events, with several instances of multiple participants present at the same fire. One PPE wipe sample for PAH analyses was not collected due to compliance issues, and three PPE wipe samples for metal analyses (i.e., one pre- and two post-fire) were omitted from the analyses due to a likely participant error. All fires were structural (i.e., involved structural components of residential or commercial buildings), except for one container fire (i.e., garbage or recycling bin).

Air samples

The air quality during on-shift fire suppression was investigated using personal air samplers worn by firefighters at the scene (Table 2-1, Figure S2-2). Personal air sample collection time averaged 67 minutes, but ranged from 10 to 420 minutes. The geometric mean total PAH concentration (± standard error of the geometric mean) was 253.1 (1.48) $\mu g/m^3$, but ranged from 8.3 to 28,600 $\mu g/m^3$ (Table 2-1). Total PAH concentrations during fires exceeded the adjusted OEL and excursion limit 45% and 31% of the time, respectively. The predominant PAHs in personal air samples were naphthalene, acenaphthylene, phenanthrene, fluoranthene, and fluorene (Table 2-1). Naphthalene was the most abundant PAH, comprising, on average, 73% of the total. However, the concentration of naphthalene never exceeded the OEL value of 52,000 $\mu g/m^3$ (Ontario Ministry of Labour, 2016). Benzo[*a*]pyrene, a known human carcinogen, comprised, on average, 1.0% of total PAHs, whereas probable and possibly carcinogenic PAHs accounted for, on average, 77% (Table S2-3).

It is interesting to compare the air PAH concentrations recorded herein with those presented in earlier studies. PAH concentrations in air from the present study contained less benzo[*a*]pyrene, but more possible and probable carcinogens (including naphthalene), compared to previously-published values for air samples collected during overhaul (i.e., activities to ensure that a fire is extinguished) (Bolstad-Johnson et al., 2000). More than half of the samples collected during the present study (i.e., from emergency fires) had higher benzo[*a*]pyrene concentrations in air compared with averages for training fires fueled with untreated wood and straw (Britz-McKibbin et al., 2016). Air samples collected during wildland, controlled burns had notably lower PAH concentrations than the on-shift air samples reported here (Robinson et al., 2008). Interestingly, Kirk & Logan (2015) noted higher average PAH concentrations in trainers' personal air samples collected during live-fire training that used particleboard, relative to the personal air samples collected in this study. However, nearly a quarter of the event-specific results reported herein exceeded those averages (Fent et al., 2017). These results show that PAH concentrations in air at emergency fires can be higher than those measured at training fires. Nevertheless, it is important to note that a study by Fent et al. (2018), which looked at PAHs in personal air samples collected at training fires fueled with household furnishings (i.e., carpeting, bed, chair, TV, etc.), showed that, depending on one's role in fire suppression, trainees experienced lower or higher personal air PAH levels compared with those presented here. More specifically, personal air

PAH concentrations for trainees assigned to command/pump roles or outside ventilation were lower than those observed here, whereas firefighters assigned to overhaul, search, and attack were drastically higher (i.e., trainees assigned to attack had median concentrations over 200-fold higher than those of this study). This comparative reduction in emergency fire PAH concentrations may be attributable to dilution by air running through the collection system while firefighters were on their way to and from a fire suppression event (i.e., compared to sample collections at training fires wherein pumps could be stopped immediately following fire suppression). Differences in PAH concentration may also be related to the duration of exposure, assignment/role, fuel, firefighter behaviors (e.g., more personnel and assignment changes at emergency fires). Overall, comparisons with previously-published values indicate that training fire studies may over-estimate (i.e., overhaul, search and attack) or under-estimate (i.e., command/pump) the PAH exposures associated with emergency fire suppression.

Concentrations of metals in personal air samples are also reported in Table 2-1. Only 10% of the air samples exceeded the adjusted OEL for lead; 7% exceeded the excursion limit. No samples exceeded the limits for antimony and cadmium. Compared to a previously reported study of air samples collected by Chicago firefighters, Ottawa firefighters experienced lower concentrations of antimony in air (i.e., 88 µg/m^3 versus average of 25.2 µg/m^3), but comparable lead concentrations (Fabian et al., 2014). Overhaul samples collected by Caux et al (2002) also had similar lead concentrations to those measured here (Caux et al., 2002). Bolstad-Johnson et al (2000) noted undetectable cadmium in air collected during overhaul activities; we only detected cadmium in 10% of personal air samples (Bolstad-Johnson et al., 2000).

Overall, variability in metal concentrations in air is large (see minimum and maximum values in Table 2-1); some values exceeded the occupational exposure limits. However, occupational exposure limits reported herein must be interpreted with caution since the proportion of the shift-time spent on emergency fire suppression will be affected by fire severity and the number of fires encountered during a shift. More specifically, municipal firefighters are rarely involved in constant fire suppression during a shift; thus, reported concentrations may overestimate the hazard. Excursion limits may be a more appropriate metric for comparison as it is a metric for short term exposures. However, if the duration of the fire suppression event exceeds the 30-minute time window for an excursion limit, this metric may underestimate total exposure. To rigorously compare firefighters’ exposures within the context of occupational exposure limits, future work should carefully consider accurate recording of exposure duration.

Collectively, these results showed that firefighters were exposed to significant amounts of airborne PAHs and selected metals during on-shift, emergency fire suppression. Since the firefighters’ occupational environment is clearly contaminated with PAHs and metals, adherence to SCBA use protocols is imperative to reduce or eliminate the risk of respiratory exposure. As noted earlier, our companion study, along with other earlier studies, revealed that dermal contact is likely a major route of firefighters’ PAH exposure (Fent et al., 2014; Britz-McKibbin et al., 2016; Keir et al., 2017). This assertion warrants further investigation.

Table 2-1 Concentrations of PAHs and metals (i.e., cadmium, antimony, and lead) in 29 personal air samples collected during emergency fire suppression. Results are reported as geometric mean (GM) and standard error (Std Error), arithmetic mean (AM), minimum (Min) and maximum (Max), in micrograms per cubic meter (μg/m^3).

Compound(s)	GM	Std Error	AM	Min.	Max.
PAHs					
Total PAHs	253.11	1.48	2725.46	8.27	28604.58
Acenaphthylene	14.57	1.58	270.50	0.09	2839.90
Acenaphthene	1.84	1.49	20.28	0.07	186.85
Anthracene	1.46	1.63	50.91	0.03	746.85
Benz[*a*]anthracene	0.91	1.48	12.59	0.02	236.05
Benzo[*a*]pyrene	1.54	1.44	10.44	0.03	133.76
Benzo[*b*]fluoranthene	1.52	1.55	17.00	0.02	218.59
Benzo[*ghi*]perylene	0.75	1.54	8.16	0.02	98.65
Benzo[*k*]fluoranthene	0.69	1.46	6.66	0.03	79.12
Chrysene	1.31	1.63	58.06	0.03	1062.72
Dibenz[*ah*]anthracene	0.27	1.29	0.70	0.02	5.58
Fluoranthene	5.95	1.53	102.17	0.11	1441.33
Fluorene	4.02	1.51	66.69	0.16	747.16
Indeno[1,2,3-*cd*]pyrene	1.07	1.47	9.34	0.04	146.36
Naphthalene	182.59	1.48	1675.80	3.95	15916.00
Phenanthrene	9.07	1.64	318.20	0.16	4543.59
Pyrene	1.97	1.79	97.94	0.02	1294.32
Metals					
Antimony	3.41	1.64	25.15	0.02	169.78
Cadmium	0.04	1.65	0.53	0.00022	6.85
Lead	0.95	1.71	33.73	0.0016	664.81

Wipe samples

We used wipe samples to assess the deposition of PAHs and metals on surfaces (i.e., skin, PPE, clothing). Post-event levels of PAHs on skin and PPE (i.e., ng per cm^2), compared to pre-event samples (i.e., collected at the beginning of a shift), increased significantly, on average by 3.3- and 5.2-fold, respectively ($p<0.001$, Figure 2-1, Table S2-4). Overall, PAHs on skin and PPE ranged from no increase (NI) to 190- and 153-fold increases, respectively. PAH accumulation on clothing increased by an average of 1.4-fold; the increase was not statistically significant. Although the detection frequency was low, the known human carcinogen benzo[*a*]pyrene was detected in all types of wipe samples (i.e., skin, PPE, and clothing). The total of probable and possible carcinogens accounted for 39%, 40%, and 33% of the total PAHs measured on skin, clothing, and PPE samples, respectively.

PAH contamination from emergency fire suppression appears to be lower than that resulting from firefighter training. Specifically, post-fire increases in PAHs on PPE were lower for the emergency responses presented herein, in comparison with those reported for PPE from firefighters exposed at training fires fueled by furniture (Fent et al., 2017). Differences are likely related to fuels and fire intensity. Additionally, reported PAH levels on instructors' PPE exposed during live-fire training were also higher, ranging from 69 to 293 ng per cm^2. Pre-fire samples were not collected in the aforementioned study, thus it's possible that the higher concentrations of PAHs on PPE are confounded by pre-event contamination (Kirk and Logan, 2015). With respect to PAHs on skin, meaningful comparisons are difficult due to differences in sample collection methods and locations on skin. Nevertheless, participants in the present study generally had lower post-event concentrations compared to those summarized in previous studies at training settings

(Laitinen et al., 2010; Britz-McKibbin et al., 2016). However, average post-event skin concentrations reported here are often higher than in training scenarios reported by Fent et al. (2017). To the best of our knowledge, this study is the first to collect emergency fire suppression samples from clothing worn under PPE. The results reveal that PAH accumulation on clothing is low compared with skin and PPE.

Post-event metal contamination on skin, clothing, and PPE was significantly higher than pre-event contamination. Average antimony on skin increased by 5.5-fold ($p<0.001$, NI to 106-fold), on clothing by 1.9-fold ($p<0.01$, NI to 19-fold), and on PPE by 1.3-fold ($p<0.05$, NI to 2.9-fold) (Table S2-4). Average lead on skin increased by 9.1-fold ($p<0.001$, NI to 719-fold), and on PPE by 3.5-fold ($p<0.001$, NI-115-fold) (Figure 2-1, Table S2-4). Post-event cadmium was always higher than pre-event sampling on skin, clothing, and PPE (e.g.,1.7-, 1.7-, 1.1-fold increases, respectively), though the differences between pre- and post-fire levels were not statistically significant. Only one other study reported PPE metal contamination (i.e., jacket, hoods, gloves), but the results were reported per g of material, thus comparisons are not possible (Fabian et al., 2014). To the best of our knowledge, this is the first study to examine post-event dermal and under-PPE clothing metal contamination in firefighters. The observed increases in metals on under-PPE clothing, which was statistically significant for antimony, suggests that these garments should be removed and laundered in a timely fashion. Moreover, standardized operating procedures should be established to ensure that all surfaces are decontaminated in order reduce exposure and internal dose. The ability of post-event surface decontamination (e.g., PPE and skin) to reduce firefighter’s exposure to metals and/or PAHs remains to be determined.

Overall, the presence of the monitored chemicals on PPE, personal clothing and skin, and the significant post-event increases in surface contamination, indicate that firefighters are significantly exposed to airborne toxicants encountered during emergency fire suppression. Moreover, significant increases in dermal contamination suggest that dermal absorption is likely a significant route of firefighters' exposures to PAHs and metals. This contention is consistent with our earlier companion study that detected a significant empirical relationship between total PAHs on skin (and air) and urinary PAH metabolites (i.e., internal dose), but failed to detect increases in biomarkers indicative of pulmonary injury (Keir et al., 2017). Nevertheless, it is important to also note that SCBA removal can result in secondary, post-event respiratory exposures to volatile or semi-volatile compounds (e.g., some PAHs and metals) (Fent et al., 2015). Indeed, this has been previously noted, i.e., premature removal of respiratory apparatus contributed to higher PAH exposures (Caux et al., 2002). Additionally, handling of contaminated clothing and PPE may also lead to elevated exposures to the monitored chemicals. The opportunities for exposure to PAHs and metals (i.e., via contact with air and contaminated surfaces), when combined with the current measurements of dermal contaminations, and our previous measurements of internal dose (e.g., urinary levels of PAH metabolites), indicate that municipal fire service organizations should actively develop policies and procedures to minimize dermal contamination, and/or post-event contact with contaminated surfaces (e.g., proper PPE use, appropriate post-event PPE handling, and skin decontamination) (Keir et al., 2017). Current research initiatives are actively evaluating the ability of on-site PPE and/or skin decontamination to reduce undesirable exposures to toxicants encountered during emergency fire suppression, and by extension, the risk of adverse health effects.

It is important to note that although sample collection at emergency firefighting events is important for real-world results interpretation, there are limitations to the study design employed in the present study. Unlike training fires, where samples can be collected immediately after fire suppression, samples for this study were collected after return to the fire station. Consequently, low molecular weight PAHs, which are typically more volatile, likely declined prior to sample collection. As such, the reported values are likely underestimating the amount of low molecular weight PAHs actually deposited on surfaces (i.e., ng per cm^2). Furthermore, although sample collection methods employed here collected 67-72% of the contamination, 28-33% remained on the surface (see Supplementary Information and Table S2-7). Thus, the wipe data presented here underestimate PAH surface concentrations within a defined confidence interval.

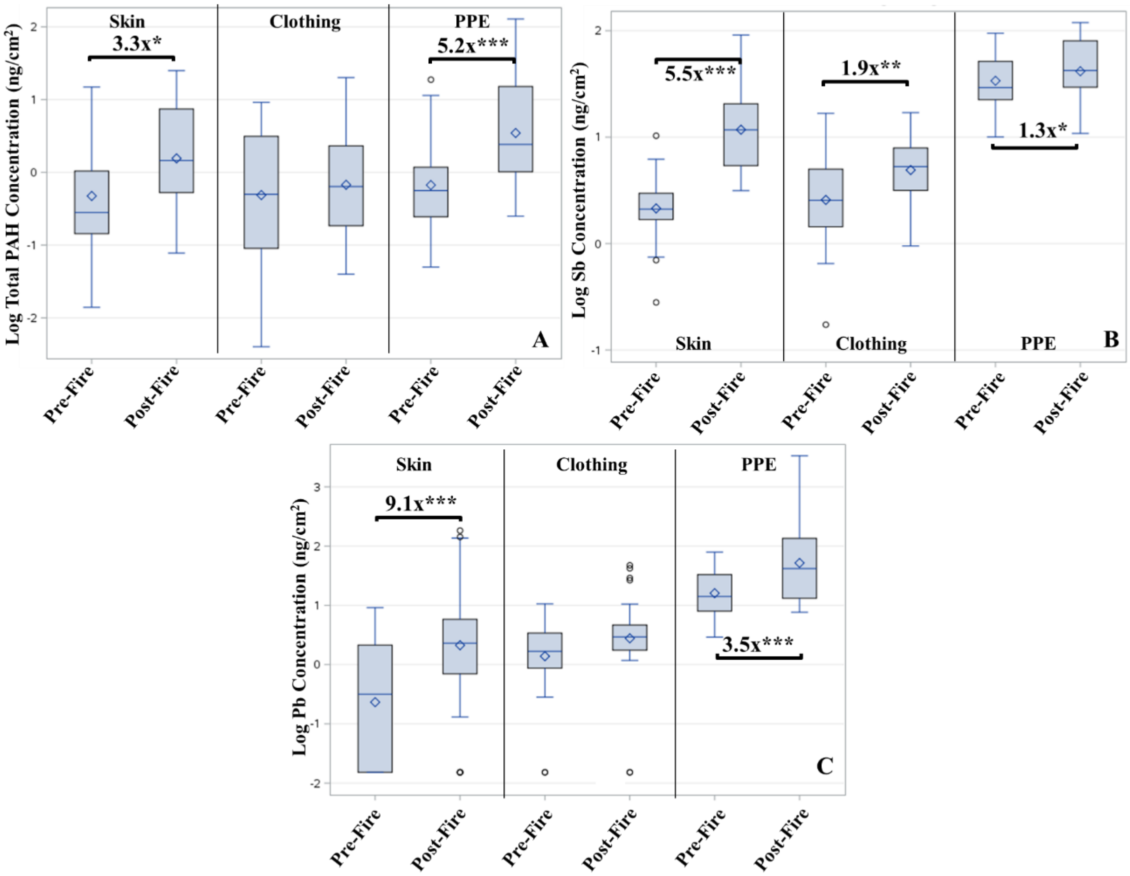

Figure 2-1 Box-plots summarizing concentrations of (A) PAHs, (B) Antimony, and (C) Lead in wipe samples collected before (FF Pre-Fire) and after (FF Post-Fire) a fire-suppression event. Box limits represent the interquartile range (i.e., 25th to 75th percentile), the diamonds represent the mean value, the solid line represents the group median, and the whiskers extend to the 5th and 95th percentiles. Circles represent outliers. * indicates a significant difference between pre and post-fire levels at $p<0.05$, ** at $p<0.01$, and *** at $p<0.001$. No significant differences in cadmium concentrations were observed and values are not shown.

Air samples from fire stations

The airborne concentrations of PAHs, antimony, cadmium, and lead were measured in vehicle bays and fire truck cabs at the three fire stations investigated, as well as at an OFS office location (i.e., reference site). Vehicle bays had significantly higher PAH concentrations compared with the office location, with geometric mean (±SE) total PAH concentrations of 95.60±1.21 and 8.20±1.0 ng/m^3, respectively (Figure 2-2, Table S2-5, $p<0.05$). In fact, vehicle bay contamination was nearly 4-fold higher than Canadian urban air (e.g., Toronto, ON, calculated annual average of 0.025 μg/m^3) (Motelay-Massei et al., 2005). Benzo[*a*]pyrene was not present in the office air samples, but was detected in 56% of the vehicle bay samples. Probable/possible carcinogenic PAHs were also more frequently detected in the vehicle bays compared to the office location (i.e., 24.8% versus 10.3%, respectively). It should be noted that the influence of outdoor, ambient air contamination on the indoor (e.g., vehicle bay) air quality data described herein cannot be overlooked since the station garage door was opened several times during the sample collection time period. Unfortunately, operational necessity precluded the ability to collect vehicle bay samples that were not periodically disturbed by door opening and truck departure/arrival. The geometric mean concentration inside truck cabs was significantly higher (i.e., 26-fold) compared to vehicle bays (Figure 2-2, $p<0.05$), and the contributions of benzo[*a*]pyrene to the total measured PAHs were higher for truck cabs compared with vehicle bays (i.e., 1.5% vs 0.02%, respectively). Similarly, the contributions of probable and possible carcinogens to the total were 65.5% versus 24.8%, respectively. To the best of our knowledge, this is the first study to note elevated concentrations of PAHs and metals inside fire truck cabs.

Metal concentrations in air showed a pattern similar to that observed for PAHs. Geometric means of antimony in vehicle bay air was significantly higher compared to the office location (i.e., Figure 2-2, 3.4-fold, $p<0.05$). Observed truck cab air concentrations were significantly higher than the vehicle bay concentrations (i.e., 50.7-fold, $p<0.05$). Antimony in air of both office and vehicle bay locations were well below those of ambient air concentrations in Ottawa for 2015 (<9 ng/m^3), and urban areas in the United States (5 to 40 ng/m^3) and Europe (1 to 10 ng/m^3) (Jensen and Bro-Rasmussen, 1992; National Research Council (US) Subcommittee on Zinc Cadmium, 1997; Environment and Climate Change Canada, 2015). However, compared to ambient Ottawa air monitored during the same time period as the present study, average fire truck cab concentrations of antimony were 117-fold higher (i.e., 0.45 versus 52.56 ng/m^3) (Environment and Climate Change Canada, 2015). Although lead in the air from vehicle bays and office locations were not significantly different, one of the truck cabs had significantly higher lead relative to the corresponding vehicle bay (Table S2-4, $p<0.05$). Office and vehicle bay air concentrations of lead were similar to those measured in Ottawa air during that time period (i.e., average of 1.1 ng/m^3); levels were higher in fire truck cabs (i.e., 13.41 ng/m^3). Concentrations in cadmium in air from all sampled locations were not significantly different from each other, and were comparable to those of Ottawa ambient air (Environment and Climate Change Canada, 2015). Overall, the observed levels of PAHs and metals in air from office and fire station (i.e., vehicle bay and truck cabs) locations were well below adjusted OELs and excursion limits. Nevertheless, the consistently elevated values in fire truck cabs warrant further attention.

Although precise delineation of factors contributing to PAH and metal contamination in truck cabs would require further study, we suspect that release of contaminants from PPE and/or contaminated gear (i.e., particles and/or off-gassed volatiles) are the major sources. Contamination is a concern because full PPE is not worn in the cab, and contamination of this area could contribute to continued exposures from earlier fires. Such secondary exposures can be reduced by cab decontamination, rigorous use of vehicle exhaust extraction systems, and proper storage of contaminated gear. Future work could investigate the effectiveness of prophylactic measures to reduce transfer of combustion-derived contaminants in stations and truck cabs (e.g., gear and PPE decontamination prior to storage and/or reuse).

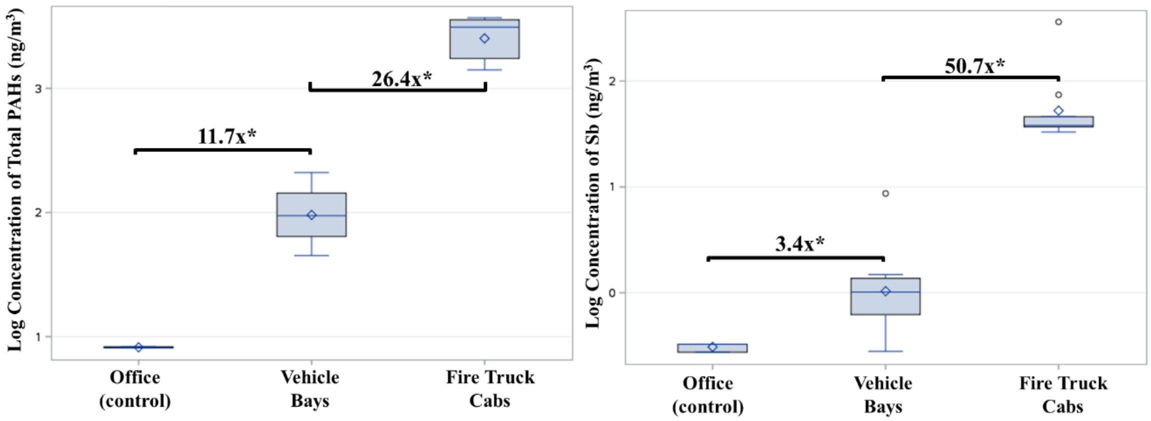

Figure 2-2 Box-plots summarizing total PAHs (left) and antimony (right) concentrations in air samples collected from control office locations, vehicle bays, and fire truck cabs. Triplicate samples were collected at each location. The box limits represent the interquartile range (i.e., 25th to 75th percentile), the diamonds represents the mean value, the solid line represents the group median, and the whiskers extend to the 5th and 95th percentiles. Circles represent outliers. The inset shows the results of a one-way ANOVA. * indicates a significant difference between locations at $p<0.05$. No significant differences in air concentrations of cadmium or lead were observed and the data are not shown.

Effectiveness of PPE decontamination

The current OFS protocol states that, weather permitting, soiled or contaminated PPE should be brushed and wetted using a fine mist, placed in a plastic bag, and dropped off for laundering on the way back to the station. Firefighters should be assigned an alternate set of PPE until laundering has been completed.[41] The effectiveness of the current laundering procedures was investigated by analyzing PPE wipe samples before and after the OFS procedural wash cycle (Table S2-6). Significant post-wash decreases in PAHs, antimony, lead, and cadmium were noted ($p<0.005$, <0.05, <0.0001, <0.01, respectively). Sets of PPE that showed a net decrease in surface contamination revealed an average laundering removal of 61% for PAHs, 55% for antimony, 97% for lead, and 90% for cadmium (Table S2-6). An estimate of PPE total surface area (i.e., pants plus jacket, Figure S2-3) was used to calculate average total removals for the compounds investigated. Values indicate removals of 166 μg of PAHs per set of PPE, including 8 μg of the known carcinogen benzo[*a*]pyrene, 912 μg of antimony, 721 μg of lead, and 26 μg of cadmium. These results should be interpreted with caution since the calculations assume uniform distribution of contamination. The efficacy of the laundering process for removal of antimony, cadmium and lead is likely related to oxidation state. Oxidized metals would be more water soluble thus, likely more readily removed. This study did not investigate the oxidation state of the analyzed metals; all results are expressed as total metal concentration. Ultimately, these results underscore the utility of PPE decontamination procedures for removing selected combustion-derived contaminants; effective decontamination should reduce the likelihood of secondary exposures mediated by contact with contaminated PPE.

The results presented herein, when interpreted in the context of our companion study and other earlier studies, indicate that firefighters engaged in emergency fire suppression are exposed to combustion-derived toxicants such as PAHs, lead, antimony and cadmium. Although this result is not surprising, the significant post-event increase in dermal contamination, and the previously-reported relationship between dermal PAH contamination and internal dose, highlights the need for further investigations. Moreover, it highlights the need to evaluate procedures intended to minimize dermal contamination, and develop intervention strategies for on-scene skin decontamination. More specifically, future work should critically scrutinize strategies to reduce event-related dermal contamination (e.g., proper PPE fitting, uniform use of flash hood, etc.), and approaches for on-scene, post-event dermal decontamination (e.g., on-scene dermal wiping). Elevated air concentrations in the truck cab and vehicle bay suggests a need for protocols to minimize cross-contamination from fire events, and/or more frequent cleaning of these areas. Current PPE decontamination protocols appear to effectively remove most of the PAHs and metals acquired during fire suppression; the efficacy highlights the utility and importance of post-event laundering. Since exposures to some PAHs and metals have been empirically and mechanistically associated with increased cancer risk, remedial or prophylactic activities designed to minimize event-related firefighter exposures can be expected to concomitantly reduce occupationally-associated cancer.

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References

American Conference of Governmental Industrial Hygienists, 2012. *TLV/BEI Guidelines*. https://www.acgih.org/tlv-bei-guidelines/tlv-chemical-substances-introduction (Accessed June 20, 2019).

Baxter, C. S., Hoffman, J. D., Knipp, M. J. et al., 2014. Exposure of firefighters to particulates and polycyclic aromatic hydrocarbons. *J Occup Environ Hyg 11*, D85–D91. https://doi.org/https://doi.org/10.1080/15459624.2014.890286.

Bolstad-Johnson, D. M., Burgess, J. L., Crutchfield, C. D. et al., 2000. Characterization of Firefighter Exposures During Fire Overhaul. *Am Ind Hyg Assoc J 61*, 636–641. https://doi.org/https://doi.org/10.1080/15298660008984572.

Brief, R. and Scala, R., 1975. Occupational Exposure Limits for Novel Work Schedules. *Am Ind Hyg Assoc J 36*, 467–469. https://doi.org/https://doi.org/10.1080/0002889758507272.

Britz-McKibbin, P., Fernando, S., Gallea, M. et al., 2016. Evaluation of Firefighter Exposure to Wood Smoke During Training Exercises at Burn Houses. *Environ Sci Technol 50*, 1536–1543. https://doi.org/https://doi.org/10.1021/acs.est.5b04752.

Caux, C., O’Brien, C. and Viau, C., 2002. Determination of firefighter exposure to polycyclic aromatic hydrocarbons and benzene during fire fighting using measurement of biological indicators. *Appl Occup Environ Hyg 17*, 379–386. https://doi.org/https://doi.org/10.1080/10473220252864987.

Daniels, R. D., Kubale, T. L., Yiin, J. H. et al., 2014. Mortality and cancer incidence in a pooled cohort of US fire fighters from San Francisco, Chicago and Philadelphia 1950-2009. *Occup Environ Med 71*, 388–397. https://doi.org/http://dx.doi.org/10.1136/oemed-2013-101662.

Edelman, P., Osterloh, J., Pirkle, J. et al., 2003. Biomonitoring of chemical exposure among New York City firefighters responding to the World Trade Center Fire and collapse. *Environ Health Perspect 111*, 1906–1911. https://doi.org/https://doi.org/10.1289/ehp.6315.

Environment and Climate Change Canada, 2015. NAPS Data Products. http://maps-cartes.ec.gc.ca/rnspa-naps/data.aspx (Accessed June 20, 2019).

Fabian, T. Z., Borgerson, J. L., Gandhi, P. D. et al., 2014. Characterization of firefighter smoke exposure. *Fire Technol 50*, 993–1019. https://doi.org/https://doi.org/10.1007/s10694-011-0212-2.

Fent, K., Evans, D., Babik, K. et al., 2018. Airborne contaminants during controlled residential fires. *J Occup Environ Hyg 15*, 399–412. https://doi.org/10.1080/15459624.2018.1445260.

Fent, K. W., Alexander, B., Roberts, J. et al., 2017. Contamination of firefighter personal protective equipment and skin and the effectiveness of decontamination procedures. *J Occup Environ Hyg 14*, 801–814. https://doi.org/10.1080/15459624.2017.1334904.

Fent, K. W., Eisenberg, J., Snawder, J. et al., 2014. Systemic exposure to PAHs and benzene in firefighters suppressing controlled structure fires. *Ann Occup Hyg 58*, 830–845.

https://doi.org/, https://doi.org/10.1093/annhyg/meu036.

Fent, K. W., Evans, D. E., Booher, D. et al., 2015. Volatile Organic Compounds Off-gassing from Firefighters' Personal Protective Equipment Ensembles after Use. *J Occup Environ Hyg 12*, 404–414. https://doi.org/10.1080/15459624.2015.1025135.

Guidotti, T. L., 2016. *Health Risks and Fair Compensation in the Fire Service*. 1st ed. Springer International Publishing, Switzerland.

Guidotti, T. L., 1993. Mortality of urban firefighters in Alberta, 1927-1987. *Am J Ind Med 23*, 921–940. https://doi.org/https://doi.org/10.1002/ajim.4700230608.

Helsel, D., 2009. Much ado about next to nothing: incorporating nondetects in science. *Ann Occup Hyg 54*, 257–262. https://doi.org/https://doi.org/10.1093/annhyg/mep092.

IARC 1989. Antimony trioxide and antimony trisulfide. *IARC Monogr Eval Carcinog Risks Hum*, 291–305

IARC, 1993. Beryllium, cadmium, mercury, and exposures in the glass manufacturing industry. *IARC Monogr Eval Carcinog Risks Hum 58*, 119-237 .

IARC, 2006. Inorganic and organic lead compounds. *IARC Monogr Eval Carcinog Risks Hum 87*, 1–529.

IARC, 2010a. Painting, firefighting, and shiftwork. *IARC Monogr Eval Carcinog Risks Hum 98*, 9–764.

IARC, 2010b. Some non-heterocyclic polycyclic aromatic hydrocarbons and some related exposures. *IARC Monogr Eval Carcinog Risks Hum 92*, 1–853

IARC, 2002. Some traditional herbal medicines, some mycotoxins, naphthalene and styrene. *IARC Monogr Eval Carcinog Risks Hum 82*, 1–556

Jensen, A. and Bro-Rasmussen, F., 1992. Reviews of Environmental Contamination and Toxicology: Environmental Cadmium in Europe. Springer, New York. https://doi.org/10.1007/978-1-4612-2890-5_3.

Keir, J. L. A., Akhtar, U. S., Matschke, D. M. J. et al. 2017., Elevated Exposures to Polycyclic Aromatic Hydrocarbons and Other Organic Mutagens in Ottawa Firefighters Participating in Emergency, On-Shift Fire Suppression. *Environ Sci Technol 51*, 12745–12755. https://doi.org/10.1021/acs.est.7b02850.

Keith, L. and Telliard, W., 1979. ES&T special report: priority pollutants: a perspective view. *Environ Sci Technol 13*, 416–423. https://doi.org/https://doi.org/10.1021/es60152a601.

Kirk, K. M. and Logan, M. B., 2015. Firefighting Instructors' Exposures to Polycyclic Aromatic Hydrocarbons During Live Fire Training Scenarios. *J Occup Environ Hyg 12*, 227–234. https://doi.org/10.1080/15459624.2014.955184.

Laitinen, J., Mäkelä, M., Mikkola, J. et al., 2010. Fire fighting trainers' exposure to carcinogenic agents in smoke diving simulators. *Toxicol Lett 192*, 61–65.

https://doi.org/https://doi.org/10.1016/j.toxlet.2009.06.864.

LeMasters, G. K., Genaidy, A. M., Succop, P. et al., 2006. Cancer risk among firefighters: a review and meta-analysis of 32 studies. *J Occup Environ Med 48*, 1189–1202. https://doi.org/10.1097/01.jom.0000246229.68697.90.

Lönnermark, A. and Blomqvist, P,. 2006. Emissions from an automobile fire. *Chemosphere 62*, 1043–1056. https://doi.org/10.1016/j.chemosphere.2005.05.002.

Motelay-Massei, A., Harner, T., Shoeib, M. et al., 2005. Using Passive Air Samplers To Assess Urban−Rural Trends for Persistent Organic Pollutants and Polycyclic Aromatic Hydrocarbons. 2. Seasonal Trends for PAHs, PCBs, and Organochlorine Pesticides. *Environ Sci Technol 39*, 5763–5773. https://doi.org/10.1021/es0504183.

National Fire Protection Agency, 2012. *NFPA 1403, Standard on live fire training evolutions*. National Fire Protection Association.

National Research Council US Subcommittee on Zinc Cadmium, 1997. *Cadmium Exposure Assessment, Transport, and Environment Fate*. National Academies Press US. https://www.ncbi.nlm.nih.gov/books/NBK233490/ (Accessed June 20, 2019).

NIOSH 2013. *Health hazard evaluation report: evaluation of dermal exposure to polycyclic aromatic hydrocarbons in fire fighters*. Cincinnati, OH. https://www.cdc.gov/niosh/hhe/reports/pdfs/2010-0156-3196.pdf (Accessed June 20, 2019).

OSHA, 2012. Coal Tar Pitch Volatiles benzene soluble fraction. https://www.osha.gov/dts/chemicalsampling/data/CH_229000.html (Accessed June 20, 2019).

Ontario Ministry of Labour, 2016. Current occupational exposure limits for Ontario workplaces required under Regulation 833. https://www.labour.gov.on.ca/english/hs/pubs/oel_table.php (Accessed June 20, 2019).

Pukkala, E., Martinsen, J., Weiderpass, E. et al., 2014. Cancer incidence among firefighters: 45 years of follow-up in five Nordic countries. *Occup Environ Med 71*, 398–404. https://doi.org/http://dx.doi.org/10.1136/oemed-2013-101803.

Robinson, M. S., Anthony, T. R., Littau, S. R. et al., 2008. Occupational PAH exposures during prescribed pile burns. *Ann Occup Hyg 52*, 497–508. https://doi.org/https://doi.org/10.1093/annhyg/men027.

Tsai, R. J., Luckhaupt, S. E., Schumacher, P. et al., 2015. Risk of cancer among firefighters in California, 1988–2007. *Am J Ind Med 58*, 715–729. https://doi.org/https://doi.org/10.1002/ajim.22466.

U.S. Environmental Protection Agency, 1994. *Method 3640A: Gel-Permeation Cleanup*. https://www.epa.gov/sites/production/files/2015-12/documents/3640a.pdf (Accessed June 20, 2019).

U.S. Environmental Protection Agency, 1996. Method 3050B Acid Digestion of Sediments, Sludges, and Soils. https://www.epa.gov/sites/production/files/2015-12/documents/3050b.pdf (Accessed June 20, 2019).

Supplementary Information

Ottawa Fire Service (OFS) PPE wash cycle

OFS’ wash cycle meets the requirements of NFPA 1851; it consists of six stages, with draining at the end of each, and a maximum temperature of 40˚C. The cycle starts with a ten-minute hot water soak and slow tumbling, followed by a 30 minute hot water wash with emulsifier and iodine. This is followed by a five-minute hot water rinse, and a 20-minute hot water wash with emulsifier and Odorstroyer™. The final stage includes a five-minute cold-water rinse, and a five-minute high-speed spin. Gear shells are separated from liners, and washed separately.

Table S2-2 Method detection limits for PAHs in wipe samples, high volume air samples (HVAS), and personal air samples (PAS).

Compound	Wipes MDL (ng/cm^2)	Air Samples MDL (ng/m^3) HVAS (Filter)	HVAS (PUF)	PAS (Filter)	PAS (PUF)
PAHs					
Acenaphthylene	0.27	N/A	0.001	N/A	0.12
Acenaphthene	2.19	0.008	0.008	1.4	0.79
Anthracene	2.20	N/A	0.008	N/A	0.71
Benz[*a*]anthracene	1.55	0.007	0.007	1.16	0.64
Benzo[*a*]pyrene	3.05	0.012	0.012	2.14	1.48
Benzo[*b*]fluoranthene	0.68	0.003	0.003	0.47	0.30
Benzo[*ghi*]perylene	N/A	0.001	0.001	0.15	N/A
Benzo[*k*]fluoranthene	N/A	0.005	0.005	0.84	N/A
Chrysene	0.83	0.003	0.003	0.61	0.40
Dibenz[*ah*]anthracene	1.37	N/A	N/A	N/A	0.88
Fluoranthene	0.44	0.001	0.001	0.26	0.19
Fluorene	3.81	0.014	0.014	2.52	1.28
Indeno[1,2,3-*cd*]pyrene	N/A	N/A	N/A	N/A	N/A
Naphthalene	7.36	0.023	0.023	4.08	1.67
Phenanthrene	0.44	0.001	0.001	0.25	0.14
Pyrene	0.31	0.001	0.001	0.16	0.14
Metals					
Cadmium	0.0087	0.0080	--	0.0017	--
Antimony	0.012	0.012	--	0.0024	--
Lead	0.019	0.019	--	0.0037	--

Cadmium, antimony, and lead were measured on filters only.

Table S2-3 Percentage of wipe samples, high volume air samples (HVAS), and personal air samples (PAS) with PAH and metal levels below detection.

	Wipe Samples						Air Samples			
	Pre-Fire			Post-Fire						
Compound	PPE	Clothing	Skin	PPE	Clothing	Skin	Office (Control)	Vehicle Bay	Fire Truck Cab	Personal Air at Fire Events
PAHs										
Acenaphthene	78.6%	82.8%	69.0%	53.6%	51.7%	48.3%	0.0%	0.0%	0.0%	0.0%
Acenaphthylene	10.7%	37.9%	13.8%	0.0%	34.5%	3.4%	0.0%	0.0%	0.0%	6.9%
Anthracene	21.4%	44.8%	20.7%	0.0%	31.0%	10.3%	0.0%	0.0%	22.2%	6.9%
Benz(a)anthracene	10.7%	27.6%	37.9%	0.0%	34.5%	6.9%	66.7%	0.0%	0.0%	20.7%
Benzo(a)pyrene	17.9%	62.1%	44.8%	7.1%	55.2%	37.9%	100.0%	44.4%	33.3%	20.7%
Benzo(b)fluoranthene	10.7%	34.5%	17.2%	3.6%	17.2%	10.3%	33.3%	0.0%	0.0%	17.2%
Benzo(g,h,i)perylene	10.7%	55.2%	20.7%	7.1%	41.4%	6.9%	66.7%	33.3%	88.9%	13.8%
Benzo(k)fluoranthene	25.0%	48.3%	48.3%	21.4%	48.3%	37.9%	100.0%	77.8%	77.8%	27.6%
Chrysene	7.1%	13.8%	6.9%	3.6%	10.3%	3.4%	66.7%	0.0%	55.6%	20.7%
Dibenz(a,h)anthracene	92.9%	96.6%	93.1%	46.4%	86.2%	82.8%	100.0%	100.0%	88.9%	44.8%
Fluoranthene	75.0%	58.6%	86.2%	39.3%	69.0%	65.5%	66.7%	0.0%	0.0%	0.0%
Fluorene	75.0%	79.3%	65.5%	32.1%	62.1%	55.2%	0.0%	0.0%	0.0%	6.9%
Indeno(1,2,3-cd)pyrene	17.9%	65.5%	48.3%	3.6%	55.2%	10.3%	66.7%	55.6%	88.9%	20.7%
Naphthalene	82.1%	58.6%	79.3%	75.0%	72.4%	75.9%	0.0%	0.0%	0.0%	0.0%
Phenanthrene	71.4%	82.8%	69.0%	25.0%	51.7%	41.4%	0.0%	0.0%	0.0%	0.0%
Pyrene	35.7%	69.0%	79.3%	21.4%	65.5%	31.0%	0.0%	0.0%	33.3%	3.4%
Metals										
Cadmium	0.0%	0.0%	0.0%	0.0%	0.0%	0.0%	0.0%	0.0%	22.2%	10.3%
Antimony	0.0%	0.0%	0.0%	0.0%	0.0%	0.0%	0.0%	0.0%	0.0%	0.0%
Lead	0.0%	0.0%	0.0%	0.0%	0.0%	0.0%	0.0%	0.0%	0.0%	3.4%

Table S2-4 Average proportions (%), and associated standard deviations (Std Dev), of total PAHs in personal air composed of the known carcinogen benzo[*a*]pyrene, the probable and possible carcinogens (IARC, 2010 & 2002), and other PAHs.

IARC Classification	**Average %**	**St Dev (%)**
Benzo(*a*)pyrene	1.0	1.3
Probable/Possible Carcinogens[a]	77.3	9.1
% Other Measured PAHs[b]	21.7	9.0

[a] Includes naphthalene, benzo(*a*)anthracene, chrysene, benzo(*b*)fluoranthene, benzo(*k*)fluoranthene, Ideno(1,2,3-*cd*)pyrene, and dibenz(*ah*)anthracene

[b] Includes acenaphthylene, acenaphthene, fluorene, phenanthrene, anthracene, fluoranthene, pyrene, and benzo(*ghi*)perylene

Table S2-5 Concentrations of PAHs and metals on skin, clothing, and PPE before (i.e., pre-fire) and after a fire suppression (i.e., post-fire) event, and the average fold change between pre- and post-fire levels. * indicates $p<0.05$, ** $p<0.01$, and *** $p<0.001$.

	Pre Fire				Post Fire				
	N	% ND	GM (ng/cm^2)	GSE (ng/cm^2)	N	% ND	GM (ng/cm^2)	GSE (ng/cm^2)	Fold Change
PAHs									
Skin	29	0.0	0.5	1.4	29	0.0	1.6	1.3	3.3***
Clothing	29	3.4	0.5	1.5	29	0.0	0.7	1.4	1.4
PPE	28[$]	0.0	0.7	1.3	28[$]	0.0	3.5	1.3	5.2***
Antimony									
Skin	29	0.0	2.14	1.1	29	0.0	11.76	1.2	5.5***
Clothing	29	0.0	2.57	1.2	29	0.0	4.91	1.1	1.9**
PPE	28[#]	3.4	33.89	1.1	27[#]	6.9	41.67	1.1	1.3*
Cadmium									
Skin	29	27.6	0.07	1.5	29	17.2	0.13	1.5	1.7
Clothing	29	13.8	0.13	1.4	29	13.8	0.15	1.4	1.1
PPE	28[#]	17.2	0.17	1.4	27[#]	20.7	0.27	1.4	1.7
Lead									
Skin	29	34.5	0.17	1.7	29	13.8	1.86	1.7	9.1***
Clothing	29	6.9	1.3	1.4	29	6.9	2.60	1.4	2.0
PPE	28[#]	3.5	16.04	1.2	27[#]	6.9	51.92	1.4	3.5***

N, number of paired samples; %ND, percent non-detects; GM, geometric mean; GSE, standard error of geometric mean.

[#] Due to operator error, three samples (1 pre-fire, 2 post-fire) were ommited from the analyses.

[$] One missing sample due to participant error.

Table S2-6 Concentrations of total PAHs, cadmium, antimony, and lead in air samples collected from an OFS office, and vehicle bays and fire truck cabs at three fire stations. Samples were collected in triplicate. Station locations (i.e., Fire station 1, 2, 3) with the same superscripted letter are not significantly different at $p<0.05$. Due to the frequency of non-detects, total PAHs values do not include benzo(*k*)fluoranthene or dibenz(*a,h*)anthracene. With the exception of two cadmium values, all other values were above the limit of detection.

Location	By Station	N	GM	Std Err	Min	Max
Total PAHs (ng/m^3)						
Office	Office^{A}	3	8.2	1.0	8.2	8.3
Vehicle Bay	Fire Station 1^{C}	3	103.3	1.2	81.5	143.5
	Fire Station 2^{C}	3	164.1	1.3	100.4	210.0
	Fire Station 3^{B}	3	51.5	1.2	44.9	64.2
	All Vehicle Bays	9	95.6	1.2	44.9	210.0
Fire Truck Cab	Fire Station 1^{D}	3	2099.8	1.3	1408.8	3113.0
	Fire Station 2^{D}	3	2735.5	1.3	1657.5	3709.2
	Fire Station 3^{D}	3	2813.7	1.3	1742.4	3582.2
	All Fire Truck Cabs	9	2528.3	1.1	1408.8	3709.2
Cadmium (ng/m^3)						
Office	Office^{A}	3	0.05	1.82	0.02	0.17
Vehicle Bay	Fire Station 1^{A}	3	0.03	1.13	0.03	0.04
	Fire Station 2^{A}	3	0.05	1.38	0.03	0.10
	Fire Station 3^{A}	3	0.03	2.39	0.01	0.10
	All Vehicle Bays	9	0.04	1.33	0.01	0.10
Fire Truck Cab	Fire Station 1^{A}	3	0.23	13.36	0.00	3.46
	Fire Station 2^{A}	3	0.22	13.61	0.00	6.23
	Fire Station 3^{A}	3	0.87	1.26	0.69	1.38
	All Fire Truck Cabs	9	0.36	2.97	0.00	6.23
Antimony (ng/m^3)						
Office	Office^{A}	3	0.31	0.33	1.06	0.27
Vehicle Bay	Fire Station 1^{B}	3	2.36	1.94	1.02	8.68
	Fire Station 2^{AB}	3	0.60	1.05	0.55	0.65
	Fire Station 3^{AB}	3	0.78	1.67	0.28	1.37
	All Vehicle Bays	9	1.04	1.38	0.28	8.68
Fire Truck Cab	Fire Station 1^{C}	3	47.12	1.26	37.04	74.24
	Fire Station 2^{C}	3	82.78	2.10	34.02	361.82
	Fire Station 3^{C}	3	37.22	1.07	33.01	41.06
	All Fire Truck Cabs	9	52.56	1.29	33.01	361.82

Lead (ng/m^3)						
Office	Office^{A}	3	1.62	10.35	2.55	0.52
Vehicle Bay	Fire Station 1^{AB}	3	3.06	1.11	2.56	3.68
	Fire Station 2^{A}	3	1.96	1.20	1.48	2.76
	Fire Station 3^{A}	3	1.67	1.64	0.64	3.28
	All Vehicle Bays	9	2.15	1.20	0.64	3.68
Fire Truck Cab	Fire Station 1^{AB}	3	21.70	4.07	1.55	185.76
	Fire Station 2^{B}	3	43.26	2.05	20.17	181.10
	Fire Station 3^{AB}	3	2.57	3.78	0.22	21.50
	All Fire Truck Cabs	9	13.41	2.08	0.22	185.76

GM, geometric mean; Std Err, standard error of geometric mean; Min, minimum; Max, maximum.

Table S2-7 Concentrations of PAHs and metals in wipe samples collected after fire suppression and before decontamination (Pre-wash), and after decontamination (Post-wash). Individual results from 12 sets of PPE are reported in ng/cm^2. Asterisks indicate statistically significant decreases at $p<0.05$ determined using paired t-test.

	PAHs*			Antimony*			Lead*			Cadmium*[#]		
Gear Set	Pre-wash (ng/cm^2)	Post-wash (ng/cm^2)	% Removed	Pre-wash (ng/cm^2)	Post-wash (ng/cm^2)	% Removed	Pre-wash (ng/cm^2)	Post-wash (ng/cm^2)	% Removed	Pre-wash (ng/cm^2)	Post-wash (ng/cm^2)	% Removed
1	1.7	3.2	-90.8	26.77	43.75	-63.42	7.11	0.42	94.14	<0.008	<0.008	0.00
2	2.0	1.3	34.5	18.04	40.23	-122.96	2.63	0.10	96.09	<0.008	<0.008	0.00
3	6.1	1.2	79.7	38.98	46.22	-18.55	21.06	0.85	95.99	0.53	<0.008	98.93
4	14.7	1.1	92.5	128.68	26.34	79.53	37.21	0.31	99.15	0.15	<0.008	96.25
5	6.7	1.3	80.4	128.80	37.98	70.51	36.99	0.11	99.70	0.26	<0.008	97.79
6	7.1	1.6	78.0	51.67	32.42	37.26	14.11	0.25	98.26	0.05	<0.008	87.60
7	2.4	1.6	33.3	52.04	34.88	32.97	14.17	0.33	97.64	0.12	0.07	40.36
8	2.1	1.1	46.4	140.35	35.60	74.64	86.84	0.67	99.23	3.07	<0.008	99.82
9	45.7	1.5	96.7	80.05	26.58	66.79	22.50	0.91	95.94	0.10	<0.008	94.14
10	3.3	2.9	14.5	29.40	23.60	19.74	9.98	0.21	97.94	<0.008	<0.008	0.00
11	3.9	1.1	71.0	63.80	34.64	45.70	79.94	0.26	99.67	8.12	<0.008	99.93
12	2.5	1.4	44.1	82.06	24.59	70.03	15.76	0.78	95.04	0.32	<0.008	98.26

[#] % removed was calculated by assigning samples below the detection limit a value equal to the detection limit divided by the square root of 2.

Wipe efficiency calculations

The efficiency of the wipe method used to collect PAHs (polycyclic aromatic hydrocarbons) on surfaces was calculated using PPE contaminated during a live fire training event. Three sequential wipes were collected from each location (i.e., back right shoulder and right knee). A new pair of gloves was used to collect each wipe. Wipes were then analysed and plotted (i.e., sequential wipe versus PAH concentration); the x-intercept was interpreted as the estimated initial concentration of PAHs on the surface. Wipe efficiency was calculated as the amount recovered from the first wipe over the estimated initial concentration. All reported values were calculated using isotopic dilution and were field blank corrected.

Table S2-8 The efficiency of the wipe collection method used to measure PAHs on surfaces. PAHs were measured on a 30 cm^2 surface and are reported in ng.

		Wipe 1	**Wipe 2**	**Wipe 3**	**Calculated wipe efficiency**
Back right shoulder	Total PAHs	553.47	398.60	61.31	67%
Right knee	Total PAHs	393.03	286.68	112.27	72%

Beginning of Shift	**During fire Event**	**Post-fire**
• Urine Sample • Skin wipes 🟦 • PPE wipes 🟩 • Under gear wipes 🟪	• Personal air sampling	• 18 hr integrated urine sample • Skin wipes 🟦 • PPE wipes 🟩 • Under gear wipes 🟪 • Post fire-event questionnaire

Figure S2-3 Illustration showing sample collection procedures for firefighter participants. The first and last panels show the locations for wipe sample collections. For the personal air samples, the white arrows in the center panel indicate the location of the pump, the connecting tubing, and the sample collection tube. Particulate material was collected on quartz filters; volatiles and semi-volatiles were collected on polyurethane foam (PUF) plugs. Photos courtesy of D. Matschke and A. Wu, used with permission. From Keir et al (2017).

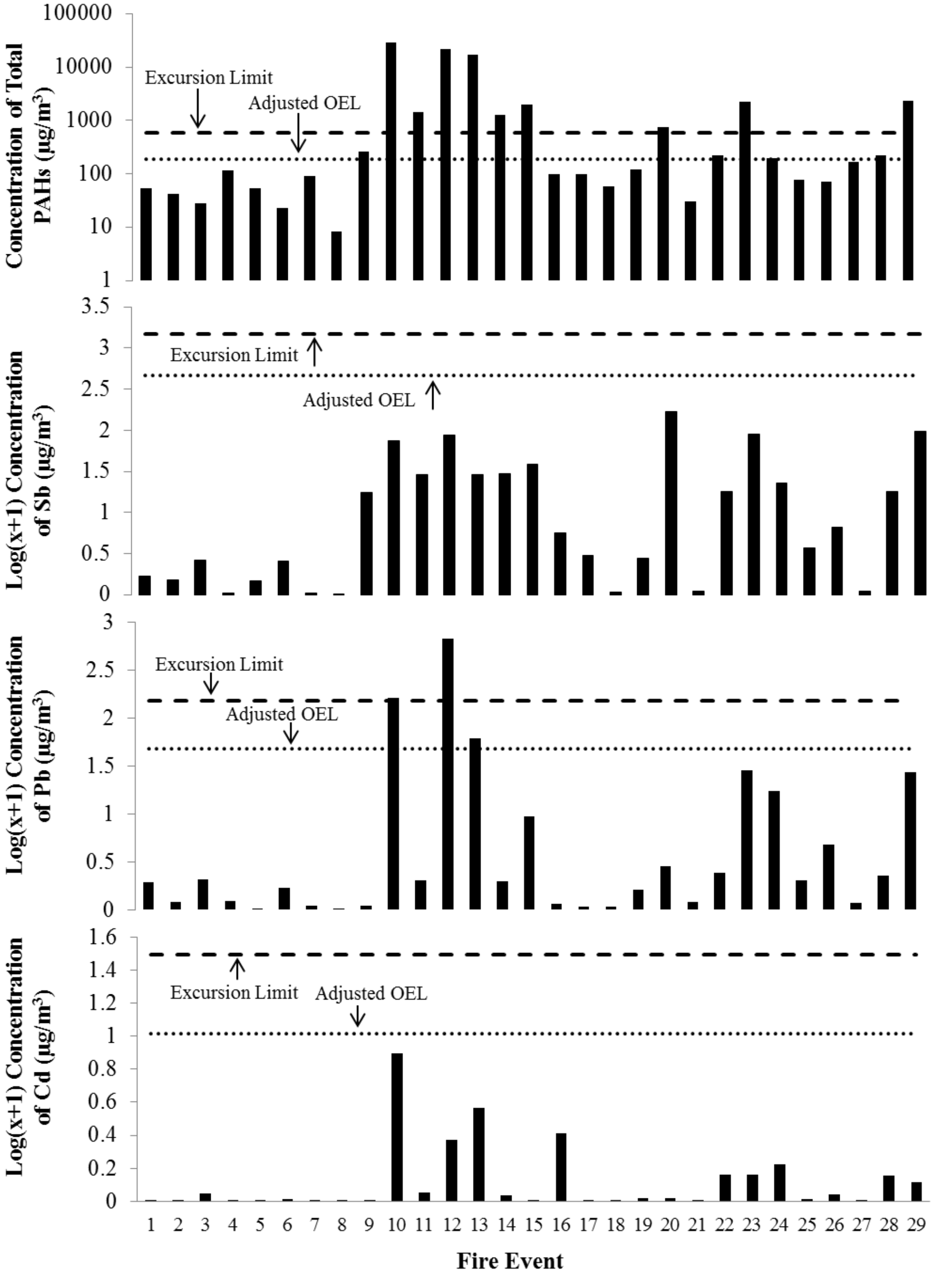
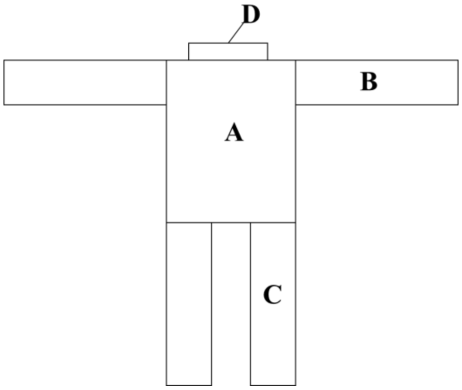


Figure S2-4 Concentrations of total PAHs, antimony (Sb), lead (Pb), and cadmium (Cd) in personal air samples collected during 29 emergency fire suppression events, with overlayed Excusion Limits and adjusted Occupational Exposure Limits (OEL).



Section	Height (cm)	Width (cm)	Area (cm^2)	Total area*(cm^2)
A – Jacket Core	85.1	63.5	5403.9	10807.7
B – Jacket Sleeves	50.8	25.4	1290.3	5161.3
C –Pant Leg	74.9	29.21	2187.8	8751.3
D -Collar	8.9	57.2	509.1	509.1
			Total	25229.4

*A = x2 (front and back)
B = x4 (front and back and two arms)
C = x4 (front and back and two arms)
D only outside is exposed

Figure S2-3: Estimated surface area of typical Ottawa firefighter PPE. Total area values were calculated using the metrics shown. The total amounts of surface-deposited PAHs and metals were calculated using wipe sample results (i.e., areal concentration values) and total surface area values.

References

IARC. Some non-heterocyclic polycyclic aromatic hydrocarbons and some related exposures. *IARC Monogr. Eval. Carcinog. Risks Hum.* **2010**, *92*, 1-853.

IARC. Some Traditional Herbal Medicines, Some Mycotoxins, Naphthalene and Styene. *IARC Monogr. Eval. Carcinog. Risks Hum.* **2002**, *82*, 1-557.

Keir, J. L. A., Akhtar, U. S., Matschke, D. M. J., Kirkham, T. L., Chan, H. M., Ayotte, P., Blais, J. M. Elevated Exposures to Polycyclic Aromatic Hydrocarbons and Other Organic Mutagens in Ottawa Firefighters Participating in Emergency, On-Shift Fire Suppression. *Environ. Sci. Technol.* ***2017,*** *51* (21), 12745–12755.

Chapter 3 Use of silicone wristbands to measure firefighters' exposures to polycyclic aromatic hydrocarbons (PAHs) during live fire training

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Statement Of Contributions

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Abstract

Firefighters experience exposures to carcinogenic and mutagenic substances, including polycyclic aromatic hydrocarbons (PAHs). Silicone wristbands (SWBs) have been used as passive samplers to measure firefighters' exposures over the course of a shift but their utility in measuring short term exposures, source of exposure, and correlations with other measurements of exposure have not yet been investigated. Here, SWBs were used to measure 16 PAH concentrations inside and outside of firefighters' personal protective equipment (PPE) while firefighting. SWBs were placed on the wrist and jacket of 20 firefighters conducting live fire training.. Correlations were made with matching data from a sister project that measured urinary PAH metabolites and PAHs from personal air concentrations from the same participants. Naphthalene, acenaphthylene and phenanthrene had the highest geometric mean concentrations in both jacket and wrist SWB (1036, 323, 179 ng/g SWB for jacket and 55.0, 4.9, and 6.0 ng/g SWB for wrist, respectively). Ratios of concentrations between the jacket and wrist SWBs were calculated as worker protection factors (WPFs) and averaged 40.1 for total PAHs and ranged between 2.8 to 214 for individual PAHs, similar to previous studies. Several significant correlations were seen between PAHs in jacket SWBs and air (e.g., total and low molecular weight PAHs, $r=0.55$ and 0.59, $p<0.05$, respectively). Few correlations were found between PAHs from SWBs worn on the wrist and jacket, and urinary PAH metabolites and air PAH concentrations. The ability of the SWBs to accurately sample PAHs was likely influenced by short sampling time, high temperatures, and high turbulence. Future work should further examine the limitations of SWBs for PAH exposures in firefighting, and other extreme environments.

Introduction

Firefighters experience elevated risks of injuries and chronic diseases; they are often exposed to stress, combustion emissions, heat, dehydration, exhaustion, and physical workload. Several studies, including extensive studies conducted by the National Institute for Occupational Health and Safety, have concluded that firefighters have higher incidence of cancer diagnoses and cancer-related deaths compared to the general populations and other occupations (Daniels et al. 2015, Pinkerton et al. 2020, Sritharan et al. 2022). Indeed, the International Agency for Research on Cancer (IARC) recently declared firefighting a Group 1 carcinogen, i.e., *known human carcinogen* (Demers et al. 2022).

Although the exact causes(s) of firefighters' increased risk of cancer and disease have not been rigorously characterized, exposures to combustion emissions are thought to be an important risk factor. Several studies have found that firefighters experience elevated external and internal markers of exposure to a variety of chemicals such as polycyclic aromatic hydrocarbons (PAHs), polybrominated diphenyl ethers (PBDEs), per- and polyfluoroalkyl substances (PFAS), polychlorinated biphenyls (PCBs), and dioxins (Chernyak et al. 2012, Park et al. 2015, Keir et al. 2017, 2020a, Trowbridge et al. 2020). PAHs are of particular interest; they are formed during combustion and are thus present at any fire suppression event. Importantly, several PAHs are known, probable or possible carcinogens (IARC 2010b, 2010a).

Firefighter biomonitoring studies have focused on examining the presence of combustion-derived chemicals, or their metabolites, in the blood, urine, saliva, and exhaled breath of firefighters after fire suppression (Fent et al. 2014, 2017, Pleil et al. 2014, Park et al. 2015, Fernando et al. 2016, Keir et al. 2017, 2020b, Wingfors et al. 2018, Santos et al.

2019). Additionally, biomonitoring studies have assessed contamination of the dermal surface (Fent et al. 2017, Wingfors et al. 2018, Sjöström et al. 2019, Keir et al. 2022). With respect to cancer risk, exposures to PAHs, most commonly assessed via the urinary concentration of metabolites, are of particular interest. For example, despite use of personal protective equipment (PPE) including self-contained breathing apparatus (SCBA), Keir et al. (2017) found a 1.9- to 5.3-fold post-suppression increase in urinary PAHs metabolite levels. Several studies suggest that dermal contact is an important determinant of total PAH exposure; moreover, more significant than respiratory system contact (Fent et al. 2014, Keir et al. 2017, Wingfors et al. 2018).

Obtaining samples to assess firefighters' personal exposures can be logistically difficult, expensive, and/or invasive. One convenient way to measure PAH exposures is by using personal passive samplers in the form of silicone wristbands (SWBs). SWBs as personal passive samplers were first described by O'Connell et al. (2014) and have since been proven useful in several PAH exposure applications, including for pregnant women, office workers, and firefighters (Dixon et al. 2018, Baum et al. 2020, Bakali et al. 2021, Mendoza-Sanchez et al. 2021, Young et al. 2021). Significant correlations have been reported between SWBs and both biomonitoring (e.g., serum, urine) and air measurements (Dixon et al. 2018, Hammel et al. 2018).

In passive samplers, such as SWBs, gas-phase chemicals partition into the sampling medium during a uptake phase; if the sampling duration is long enough, equilibrium is reached. In the kinetic or linear region of the uptake phase, the mass collected is proportional to the concentration in the air and the time of exposure. Molecular diffusion (i.e., Fick's first law of diffusion) is a slow process and, consequently, sampling rates of SWBs are low, and longer sampling duration is required in order to obtain adequate sensitivity. Due to time constrains, SWBs usually operate in the linear region.

SWBs can be used to estimate environmental contamination, such as in air, using a linear uptake model, with partitioning coefficients ($K_{\text{SWB-Air}}$) calculated as ratios between SWBs and air concentrations. $K_{\text{SWB-Air}}$ are useful to investigate a compound's distribution amongst the two phases (i.e., silicone and air) at a given point in time. True $K_{\text{SWB-Air}}$ values are reported when compounds reach equilibrium. However, to provide information on whether a compound is approaching equilibrium between the two phases, $K_{\text{SWB-Air}}$ values can still be calculated prior to equilibrium (Anderson et al. 2017).

Sampling rates, R (m^3 day^{-1}), of passive samplers are often calculated by comparing passive sampler concentrations with those obtained by active samplers to provide an estimate of amount of environmental matrix measured (i.e., m^3 of air over a period of deployment) (Čáslavský et al. 2004). Sampling rates are useful as they can be used to estimate environmental contamination using SWB data. For example, in a firefighting scenario, sampling rates of SWBs may be useful to estimate a time-averaged air concentration, i.e., by multiplying R by the time the sampler was deployed. If SWBs reach saturation points (i.e., equilibrium with the environmental matrix such as air), correction factors must be applied (Pozo et al. 2009).

In an effort to better understand firefighters' environmental exposures to targeted chemicals, several studies have been conducted using SWBs. A pilot study by Baum et al (2020) showed that SWBs can be used to evaluate FFs' exposures to PAHs, with elevated PAHs being measured in SWBs worn by firefighters who attended a fire suppression event over the course of a 24-hour shift (Baum et al. 2020). Similarly, Caban-Martinez et al. (2020)

also found elevated PAH concentrations in SWBs worn by Dominican Republic firefighters who attended a fire while on duty (Caban-Martinez et al. 2020). SWBs have also been used to assess PAH deposition in various safety zones of a fire (i.e., hot, warm, or cold zones); moreover, differences in PAHs generated by different types of fuels. Warm and cold zones, thought to be relatively safe, had measurable amounts of PAHs, and elevated PAHs were measured in fires fueled by biomass and wood compared to propane (Bakali et al. 2021). Using a modified approach, Poutasse et al (2020) used silicone "dog-tags" to show that firefighters are exposed to PAHs, including 18 PAHs not previously investigated in firefighter exposure studies (Poutasse et al. 2020). Recently, Levasseur et al. (2022) used SWBs to assess levels of 134 chemicals; flame retardants and PAHs were associated with firefighting. A pilot study by Bonner et al. (2023) used SWBs to assess levels of dermal protection by a novel PPE configuration; the study found that PPE with reduced gaps had lower PAH penetration. However, these earlier studies failed to acknowledge and investigate the influence of short sample duration, high temperatures, and unpredictable air turbulence that has previous been shown to affect SWB passive sampling (Harner et al. 2013, Anderson et al. 2017, Donald et al. 2019). Furthermore, no study has used SWBs to assess firefighters' PAH exposures during live fire training, nor attempted to compare SWB results with the active air sampler results and the results of urinary PAH metabolite analysis. This study employed SWBs to examine firefighters' PAH exposures during live fire training; results are compared to matched personal air PAH concentrations and urinary PAH metabolite analyses. The results will be used to further examine firefighters' exposures to PAHs and aid in future design of studies using SWBs.

Materials and Methods

Recruitment

Research ethics approval was obtained from the University of Ottawa Research Ethics Board (i.e., H-08-18-867) and Health Canada's Research Ethics Board (i.e., REB 2018-0020). Participants were recruited from the Ottawa Fire Services (OFS) and the Canadian Armed Forces. All participants were enrolled in OFS live fire training. The inclusion criteria were that study participants were non-smokers, did not live with smokers, and agreed to avoid consumption of charbroiled foods and non-occupational exposures to combustion sources for seven days before the training event, and one day after the training event. Informed consent forms and general lifestyle questionnaires were completed by all subjects. Details of the recruitment process can be found in Keir et al. (2022).

During the training fires, 20 firefighters wore two SWBs: one SWB was placed on the individual's non-dominant wrist and the other attached to the radio velcro loop on the chest of the bunker gear. Research staff wearing nitrile gloves removed the SWB from their respective bags and placed the SWB on the wrist and jacket of the firefighter immediately prior to entering the container where the live fire was occurring. After the fire, new nitrile gloves were donned; the SWBs were retrieved and placed back in their original bags. Field and travel blanks were collected at the time of exposure. All samples were placed in coolers with ice packs before transportation for analyses.

As part of the Keir et al. (2022) study, the participants also provided urine samples pre- and post-fire; pre-fire samples were collected approximately one hour before the live fire training scenario began and an integrated 18-hr post-fire urine sample was collected after the fire. At least one participant in each fire event wore a personal air sampler such that active air samples were collected from each training fire (n=14). 16 PAHs were measured in the SWB and active air samples, and PAH metabolites were measured in urine. Osmolality was used to correct for urinary dilution. PAHs in active air samples were analysed via GC-MS and 19 urinary PAH metabolites via GC-MS/MS, as described in Keir et al. (2022). PAHs analysis of SWBs were conducted as described below. All samples were stored in a cooler during transportation to a -20°C freezer where they remained until analysis.

Chemicals and standards

A 16 PAH native standard mix and a 16 PAH deuterated internal standard mix were purchased from Wellington Laboratories (Hamilton, ON, Canada). Terphenyl d-14 was purchased from Cambridge Laboratories (Massachusetts, USA). Details of the standards can be found in the supplementary information.

Silicone wristbands

Pre-cleaned silicone wristbands were obtained from MyExposome (Corvallis, OR, USA). After deployment to the training fires (or not, for the blanks), all SWBs were kept at -20ºC in their original sealed bag until extraction and analysis.

Sample preparation

SWBs were extracted by accelerated solvent extraction using an ASE 200 (Dionex, ON, Canada) at 1500 psi and 70°C with hexane:acetone (70:30), for 3 cycles. ASE extraction cells (22 mL) were pre-packed with a glass fiber filter and 1 g of each 3% H_2O-deactivated alumina, silica, and florisil followed by anhydrous Na_2SO_4. Another glass fiber filter was inserted into the cell and headspace was filled with hydromatrix (ThermoFisher Scientific, MA). SWBs were spiked with 10 µL of internal standard mix and allowed to dry before topping with a final glass fiber filter. Extracts were collected in ASE glass vials (60 mL) and evaporated to 2 mL at 40ºC in a Turbovap Classic LV (Biotage, NC, USA) under a gentle stream of nitrogen. This was followed by two 10 mL solvent exchanges with hexane, evaporation to 1 mL final volume, and spiked with recovery standard. Samples were analysed using a TSQ Quantum XLS Ultra™ Gas Chromatography-triple quadrupole mass spectrometer (GC-MS/MS) (ThermoFisher Scientific, MA). Details of the instrument settings can be found in Table S3-1. Method blanks, field blanks, and spiked SWBs were analyzed alongside the collected samples.

Statistical Analysis

PAHs with greater than 80% of samples below the detection limit were omitted from the analyses and PAH sum determinations. If more than 80% of the samples had detectable concentrations, values below the detection limit were replaced with the limit of detection divided by the square root of 2. If 20% to 80% of the samples were above the detection limit, non-detects were replaced with values imputed using NDExpo Version 1.0 (http://expostats.ca/site/app-local/NDExpo/). NDExpo imputes values using robust regression on order statistics (Helsel 2009). High and low molecular weight PAHs were calculated using the sum of PAHs with 4 or more rings or 3 or fewer rings, respectively. Data were analyzed using SAS OnDemand for Academics (SAS Institute, Cary, NC, USA) and Microsoft Excel. A paired t-test was used to investigate the effect of SWB deployment location, i.e., wrist versus jacket. Correlation analysis was used to investigate the correspondence between PAHs levels in SWBs on the wrist and jacket, and (i) PAHs in air, and (ii) urinary PAH metabolite levels. Correlation analyses employed Pearson product-moment correlation coefficients determined using PROC CORR in SAS. Data were log transformed to equalise the variance across the range of observations. Statistical significance was defined as $p < 0.05$.

Worker protection factors (WPFs) estimate how protected a worker is from compounds found in their environment; they are calculated as the ratio of the concentration of a substance outside of the personal protective equipment (PPE) to the concentration underneath the PPE (Wingfors et al. 2019). For each participant, WPFs were calculated using the concentration of the PAH in the jacket SWB divided by the wrist SWB. Importantly, wrist SWBs were covered by the Bunker gear jacket.

Silicone-air partitioning coefficients ($K_{\mathrm{SWB\text{-}Air}}$) are often calculated to investigate the interactions between PAHs in air and SWBs; they are calculated as the ratio between the PAH in SWB and air:

Equation 1:

$$K_{\mathrm{SWB-Air}} = \frac{C_{SWB}}{C_{Air}}$$

Where $K_{\mathrm{SWB\text{-}Air}}$ is the SWB-air distribution coefficient (L kg^{-1}), C_{SWB} is the amount of PAH in the SWB (ng kg^{-1} SWB), and C_{Air} is the concentration of PAHs measured using the personal air samplers (ng L^{-1}).

Sampling rates (R_{SWB}) were calculated to represent the volume of air sampled by the passive sampler using the following equation derived from Čáslavský et al. (2004):

Equation 2:

$$R_{SWB} = \frac{C_{SWB} x\ m_{SWB}}{C_{Air} x\ t}$$

Where R_{SWB} is the sampling rate (m^3 day^{-1}), C_{SWB} is the amount of PAH in the SWB ($ng\ g^{-1}$ SWB), m_{SWB} is the mass of the SWB in g, C_{Air} is the concentration of PAHs measured using the personal air samplers in $ng\ m^{-3}$, and t is the length of time the SWB was deployed (i.e., sampling time) in days. $K_{SWB\text{-}Air}$ and R_{SWB} were only calculated for jacket SWBs as they were in direct contact with air whereas wrist SWBs were protected by PPE.

Temperatures within the fire environments examined herein varied due to the dynamic nature of the combustion; they were approximated using values from Randall (2020), who measured temperatures in training fires conducted at the same facility with the same fuel. The estimated temperature for this study was based off measurements from in the middle of the shipping container 75 cm from the floor, near where the jacket would have been located. The estimated temperature was 100°C.

Results

A total of 20 participants deployed SWBs on the wrist and jacket (i.e., 40 SWBs total). On average, the SWBs were worn between 39 and 63 min; the average was 53 min.

Table 3-1 PAH concentrations in silicone wristbands (SWBs) worn by firefighters during a training fire, and Worker Protection Factors (WPF). SWB units are nanogram per gram SWB and WPFs are the unitless ratios of PAH concentrations in jacket SWBs to wrist SWBs.

PAH	Wrist					Jacket					WPF
	DF	GM	SE	Min	Max	DF	GM	SE	Min	Max	Mean
Naphthalene	100%	55.0	13.6	4.4	369.4	100%	1036	144.4	285.7	3313	41.6
Acenaphthylene	100%	4.9	1.4	0.6	54.9	100%	322.8	44.4	108.9	1254	213.8
Acenaphthene	70%	0.9	0.2	0.2	5.2	100%	40.3	5.6	11.9	130.5	79.4
Fluorene	100%	2.2	0.4	0.4	12.2	100%	92.6	10.9	31.5	264.7	70.3
Phenanthrene	100%	6.0	1.3	0.6	33.8	100%	178.5	25.5	42.8	479.4	68.3
Anthracene	65%	0.6	0.2	0.1	6.8	100%	44.6	6.9	10.7	175.5	123.8
Fluoranthene	70%	2.2	0.3	0.8	7.0	100%	25.5	5.1	2.6	118.3	16.8
Pyrene	75%	1.4	0.3	0.2	5.6	100%	20.4	4.0	2.2	99.4	25.0
Benzo[*a*]anthracene	5%	NA	NA	NA	NA	85%	1.7	0.5	0.2	14.1	NA
Chrysene	10%	NA	NA	NA	NA	80%	2.1	0.5	0.4	18.6	NA
Benzo[*b*]fluoranthene	20%	0.3	0.1	0.0	2.7	65%	1.0	0.2	0.1	7.0	6.6
Benzo[*k*]fluoranthene	10%	NA	NA	NA	NA	35%	0.4	0.1	0.0	4.4	NA
Benzo[*a*]pyrene	30%	0.5	0.1	0.1	2.4	55%	1.1	0.3	0.1	9.7	5.5
Indeno[1,2,3-cd]pyrene	15%	NA	NA	NA	NA	55%	0.5	0.2	NA	NA	NA
Dibenzo[*a*,h]anthracene	20%	0.1	0.0	0.0	3.6	15%	NA	NA	0.0	5.5	NA
Benzo[*g*,*h*,*i*]perylene	30%	0.3	0.1	0.0	3.8	60%	0.7	0.2	0.0	7.2	2.8
Total LMW PAHs		73.2	16.7	7.3	482.3		1733.2	232.6	531.4	5591.5	46.9
Total HMW PAHs		6.1	0.7	2.3	20.8		56.3	11.3	5.9	232.8	13.7
Total PAHs		82.7	16.9	13.9	503.1		1796.0	242.7	537.4	5824.3	40.1

DF=detection frequency, GM= geometric mean; SE = standard error, Min= minimum, Max= maximum, NA= not available due to >80% of samples being below the detection limit, WPF = worker protection factor, i.e., ratio of jacket PAH concentration to wrist PAH concentration.

All the sixteen PAHs targeted in the study were detected in at least two samples (Table 3-1). PAHs with >80% of samples below the detection limit were omitted from analyses and PAH sum calculation (i.e., benzo[*a*]anthracene, chrysene, benzo[*k*]fluoranthene, and indeno[1,2,3-*cd*]pyrene for wrist SWB and dibenzo[*a*,*h*]anthracene for jacket SWB). Naphthalene, acenaphthylene and phenanthrene had the highest geometric mean concentrations in both jacket and wrist SWB (Table 3-1). The three PAHs with highest concentrations in SWBs were the same as those found in personal air samples from the same training fires (Keir et al. 2022). Compared to low molecular weight (LMW) PAHs, high molecular weight (HMW) PAHs were 12- and 30-fold lower in the wrist and jacket SWB, respectively. Using a paired t-test, the results showed significantly higher PAH concentrations in jacket SWBs compared to wrist SWBs for all PAHs except benzo[*a*]pyrene ($p<0.01$). However, the results also revealed significant correlations between the levels of several PAHs on the wrist and jacket SWB (Table S3-5); these results mainly pertain to HMW PAHs.

Worker protection factors (WPFs), which are the unitless ratio of concentrations of a compound outside to inside worker PPE ranged from 2.8 to 213.8 (Table 3-1). Higher MW PAHs tended to have lower WPFs. For example, WPFs for benzo[*b*]fluoranthene, benzo[*a*]pyrene, and benzo[*g*,*h*,*i*]perylene ranged between 2.8 and 6.6, whereas lower MW PAHs ranged between 25.0 and 213.8. In fact, for the PAHs examined, there was a significant, inverse correlation between WPFs and molecular weight ($r^2=0.39$, $p<0.05$, Figure 3-1A). WPFs were also compared with those from Bonner et al. (2023), who calculated WPFs from SWBs placed under and in the surrounding environment of standard and one-piece PPE (Table S3-2). Similar patterns were seen, with lower MW PAHs having higher

WPFs than higher MW PAHs, but Bonner et al WPFs were consistently higher than those noted herein. More specifically, WPFs for LMW and HMW PAHs were 11- and 8-fold higher for standard PPE and 16- and 82-fold higher for one-piece PPE, respectively (Bonner et al., 2023).

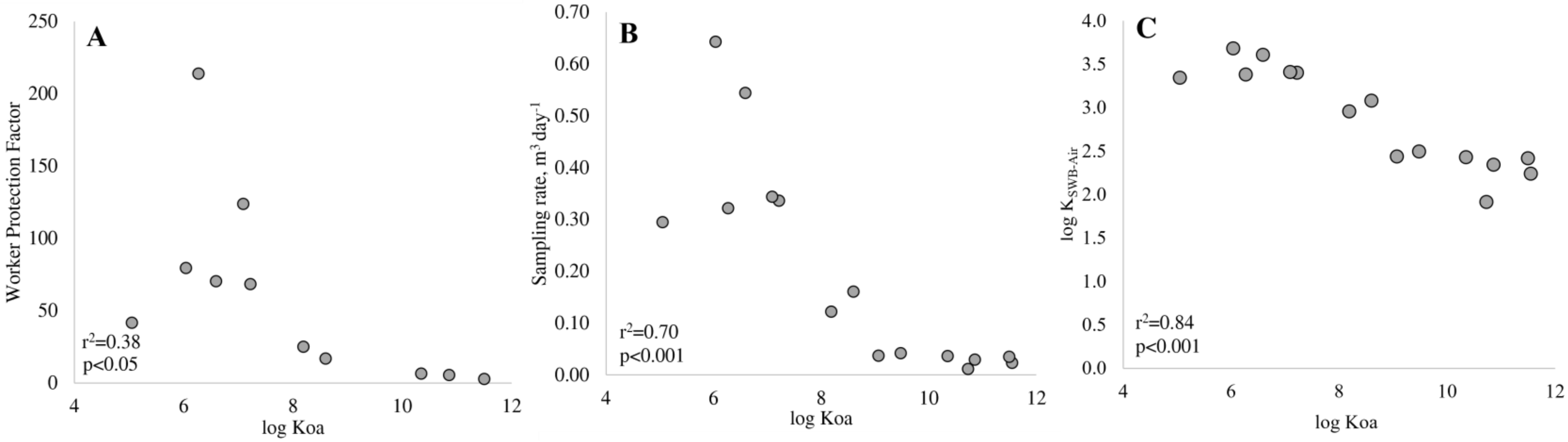

Figure 3-1 Worker Protection Factor (WPF, unitless) (A), sampling rate (R, m^3 day^{-1}) (B), and log $K_{\text{SWB-Air}}$ (L kg^{-1}) (C) compared to log octanal-air partition coefficient (K_{oa}). WPF, sampling rate and $K_{\text{SWB-Air}}$ were calculated using PAH levels on jacket SWBs. Data for certain PAHs are missing due to values for one or more of the variables being below detection.

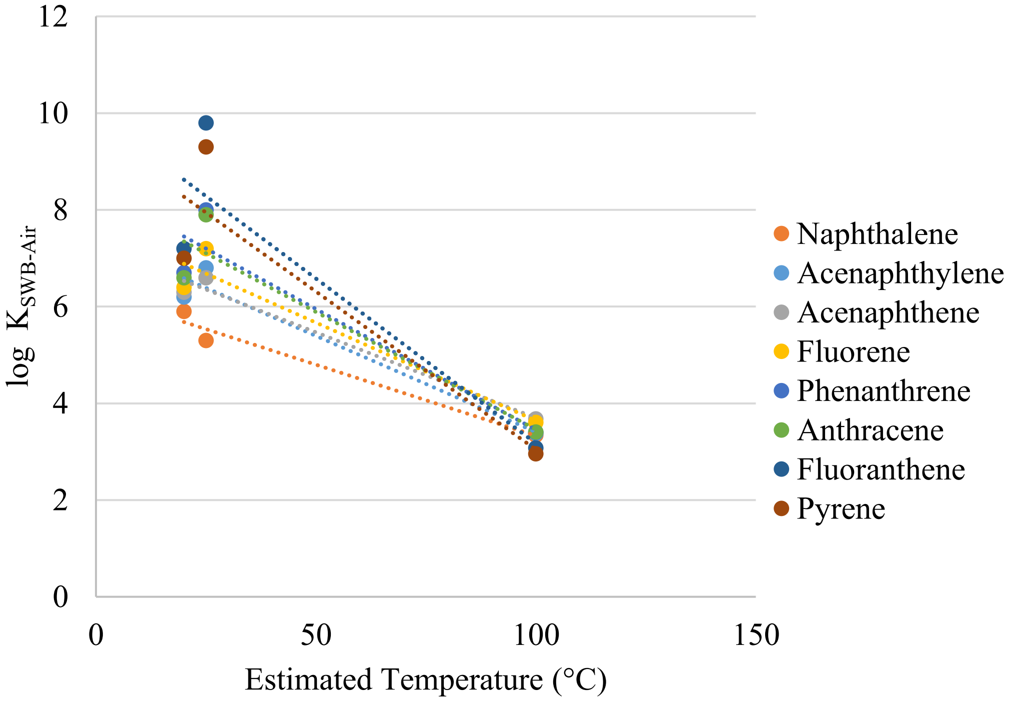

Figure 3-2 Average log $K_{SWB-Air}$ from Anderson et al. (2017) estimated at 20°C, Donald et al. (2019) estimated at 25°C, and here estimated at 100°C based off temperature estimates from Randal (2020). Only PAHs with data from all three studies were included.

To investigate the ability of SWBs to capture environmental PAHs, correlations between PAHs on jacket SWBs and personal air PAH levels were investigated (Table S3-6). Grouped PAHs (i.e., LMW, HMW, and total) from jacket SWBs correlated with nearly all air PAH levels; however, correlations between individual PAHs were mainly between LMW PAHs from jacket SWBs and a range of PAHs in air.

Correlations between wrist SWBs and urinary PAH metabolites were used to determine SWBs' ability to predict internal dose, i.e., level of urinary PAH metabolites. The only significant correlation was between post-fire total urinary PAH metabolites and benzo[*g*,*h*,*i*]perylene in the wrist SWBs (r=0.52, p=0.03). Additional analyses examined correlations between urinary PAH metabolite levels and jacket SWB levels. The latter analyses were employed to determine if PAH levels on SWBs exposed to environmental levels of PAHs were empirically related to urinary PAH metabolites (Table S3-7). The results revealed that post-fire ΣOH-naphthalenes was significantly correlated with chrysene and benzo[*k*]fluoranthene on the jacket SWB (r=0.50 and r=0.51, respectively, p<0.05 for both). Post-fire total urinary PAH metabolites correlated with benzo[*a*]anthracene, chrysene, and benzo[*k*]fluoranthene on jacket SWB (r=0.54, 0.59, 0.51, respectively, p<0.05).

Additional analyses did not reveal any statistically significant relationships between PAH levels on wrist or jacket SWBs and variables pertaining to participants' age, height, weight, or gender. Additionally, there was no significant relationship between PAH levels and the amount of time the SWB was worn at the fire event (data not shown).

$K_{\text{SWB-Air}}$ were strikingly lower than those previously reported (Table S3-8). Specifically, compared to Anderson et al. (2017) and Donald et al. (2019), who deployed SWBs for 48h and 27 days, respectively, average log $K_{\text{SWB-Air}}$ of jacket SWBs for LMW PAHs were 1.8- and 2.0-fold lower, and for HMW PAHs, were 2.3- and 4.8-fold lower. The results also revealed an inverse relationship between log $K_{\text{SWB-Air}}$ and log K_{oa} (Figure 3-1B). Sampling rates (R) were also significantly correlated with log K_{oa} (Figure 3-1C). Relatedly, HMW PAH sampling rates were approximately half that of those determined for LMW PAHs.

Discussion

This is the first study to assess the ability of SWBs to assess firefighters' exposures to PAHs during training firefighting; matching samples were collected using active air monitoring and urinary PAH metabolite analyses. The results obtained show that PAHs could be detected on exposed SWBs, although several HMW PAHs were frequently below detection limits. Comparisons with active air samples and urinary PAH metabolite concentrations showed significant correlations between jacket SWBs and air samples, but limited correlations for all other comparisons.

Although the results indicate that SWBs were able to assess levels of PAHs in the fire environment, and by extension firefighters' PAHs exposures, several factors likely influenced the ability of SWBs to reliably assess PAH exposures in the investigated scenario. Indeed, it's well documented that passive sampling of PAHs in the atmosphere is complicated by vapor-particle partitioning, which is dependent on several factors that are discussed below (i.e., sampling time, temperature, air velocity). Effective interpretation of the results obtained, and their implications for exposure assessment, requires incremental consideration of these factors.

First, the relatively short sampling time likely had a profound effect on the observed SWB PAHs. Depending on the phase partitioning between PAHs in the air and SWBs, PAHs of various molecular weights, and relatedly, log K_{oa}, will require different amounts of time to absorb into the SWB. O'Connell et al. (2021) reported that PAH concentrations of phenanthrene, pyrene, and fluorene were still in the linear uptake phase even after 15 days; PAHs with higher K_{oa} (i.e., HMW PAHs) would require even more time to equilibrate. Furthermore, previously reported $K_{\text{SWB-Air}}$, assumed to be at equilibrium, were significantly higher than the distribution coefficients reported herein, more so for the higher MW PAHs. This further illustrates the SWBs' departure from equilibrium in the present study (Anderson et al. 2017, Donald et al. 2019). Thus, the period of exposure of the SWBs herein may not have been enough to collect sufficient HMW PAHs to be detectable given their lower sampling rates, thus explaining the low detection rates of HMW PAHs in a highly contaminated environment.

Second, environmental temperatures associated with the jacket and wrist SWBs were substantially higher than the ambient conditions (~20-25°C) of previous SWB exposure assessment studies. The jacket SWB was subjected to an estimated average temperature of ~100°C; however, the external temperature very likely went up to >175°C (Randall 2020). The wrist SWB was likely subjected to temperatures of ~38°C, as measured by Horn et al. (2018) on firefighters' arm surfaces during a staged fire. These higher temperatures likely influenced PAH absorption by reducing deposition rates.

To investigate the influence of temperature on partitioning between the silicone and air phases, relationships between $K_{\text{SWB-Air}}$ and temperature were investigated by comparing log $K_{\text{SWB-Air}}$ here with those reported by Anderson et al. (2017) and Donald et al. (2019). The former study examined PAH concentrations from SWBs and active personal air samplers worn by 22 women in New York City for 48hr. The latter study reported PAH concentrations from SWBs and passive air samplers deployed near turf sport fields for 27 days. Only PAHs with data available from all three studies were included. Temperatures from Anderson et al. (2017) and Donald et al. (2019) were estimated using reported conditions (i.e., ~20°C and up to ~25°C, respectively). As noted, the log $K_{\text{SWB-Air}}$ values calculated here were lower than those indicated in the other studies, i.e., 1.9 to 3.7 compared to 5.3 and 13.8 in studies conducted at temperatures near that of typical indoor air (Anderson et al. 2017, Donald et al. 2019). Differences in the coefficients are also likely influenced by the differences in deployment time and consequently, departure from equilibrium as previously discussed. Indeed, calculation of $K_{\text{SWB-Air}}$ assumes equilibrium has been reached, thus comparisons of $K_{\text{SWB-Air}}$ with other studies of longer deployment times must be interpreted with caution. Nonetheless, the negative linear relationship between $K_{\text{SWB-Air}}$ and temperature seen here (Figure 3-2) has also been reported by Donald et al. (2019). Donald et al. (2019) had previously reported 2-fold decreases in $\mathrm{K}_{\text{SWB–Air}}$ with every 10°C increase in temperature, but the relationship between $\mathrm{K}_{\text{SWB–Air}}$ and temperature noted here was decidedly steeper (Figure 3-2). The differences between the two studies are likely due to the magnitude of temperature range examined, i.e., Donald et al. calculations were based on a several degree temperature change whereas analyses conducted herein spanned ~80°C, as well as differences in sampling time. The differences in $\mathrm{K}_{\text{SWB–Air}}$ between all three studies are exacerbated by increasing molecular weight (Table S3-8); this pattern is likely a result of the short deployment times and slow deposition kinetics for HMW PAHs in the present study. Overall, the lower log $K_{\text{SWB-Air}}$ values here suggest that PAHs are less likely to be absorbed into the SWBs examined. That said, the presented analyses must be interpreted with caution since only three temperatures were available for analysis of the relationship between $K_{SWB-Air}$ and temperature; moreover, constant temperature was assumed in each instance. In addition to the fact that temperature increases reduce the absorption of PAHs in SWBs, it is important to note that higher temperatures also increase skin absorption rates (Akomeah et al. 2004). Specific to high temperature environments, this nullifies a previous assumption that the relatedness of absorption capacities between SWBs and skin make SWBs good indicators of dermal exposure (Hammel et al. 2018). In other words, the temperature of the environments that firefighters face causes differences in absorption capacities between SWBs and skin, lessening the notion that absorption of PAHs to a SWB is equivocal to dermal absorption.

Turbulence is another factor within the fire environment that may affect the ability of SWBs to assess ambient PAHs levels. Indeed, gas velocities in a staged fire scenario have been reported to be up to >2 m s^{-1} (Kerber, 2010), 10-fold higher than ambient air speeds likely encountered during previous SWB studies (Arens et al. 2020). Higher wind speed has been shown to increase linear-phase sampling rates by lessening the boundary layer surrounding a passive sampler, and suggests an air-side controlled uptake mechanism (Harner et al. 2013). Simultaneously, a turbulent environment may reduce the ability of particles, and any PAHs bound to them (i.e., HMW PAHs), to settle and allow the PAH to be absorbed by the SWB, thereby adding to reasons why HMW PAHs are difficult to sample using SWBs in a live fire scenario.

Overall, it is likely that temperature and turbulence had a significant influence on the ability of SWBs to accumulate ambient PAH levels, resulting in an underestimation of PAHs, particularly the HMW PAHs.

The nature of the SWB deployment may also have influenced the results obtained. The placement of the wristbands may have affected results, with the inside of the wrist SWB having less surface area due to being partially covered by the wrist itself. In contrast, the jacket SWB was hung from a loop on the external jacket surface, and thus had more exposed surface area. This may have underestimated the concentration of PAHs under the personal protective equipment (PPE) compared to outside the PPE. Furthermore, this difference in SWB placement may partly account for the lack of correlation between SWBs results and internal markers of PAH exposure (i.e., urinary PAH metabolites). More specifically, the location of the SWBs may not have permitted accurate assessment of PAH dermal contact, which has been highlighted as an important determinant of firefighters' PAH exposures (Fent et al. 2017, Keir et al. 2017, Wingfors et al. 2018). Indeed, it has been shown that other dermal areas of the body that can be more exposed (e.g., neck and chest) (Bonner et al. 2023); it might be more suitable to employ silicone dog tags that can be worn around the neck (Poutasse et al., 2020). Alternatively, the lack of correlations between ambient air and SWB PAH levels, and between urinary PAH metabolite and SWB PAH levels, may indicate that dermal exposure is not a predominant route of firefighters' PAH exposures. In fact, Andersen et al. (2017) noted that firefighters were most exposed to ambient particulate matter during situations where they doffed their self-contained breathing apparatus (SCBA) when they were considered to be in "safe zones" (i.e., away from the fire). Firefighters' exposures to PAHs are likely an interplay of various routes of exposure.

While recognizing the effects that a firefighting environment may have on SWBs' ability to sample PAHs, it is important to note that SWBs can still provide important information about firefighters' exposure potential. In fact, there have been several previous studies that have used SWBs to assess firefighter exposures during other types of fire suppression (e.g., on-shift). Results were similar, albeit for studies with longer sampling times. SWBs worn in the study by Baum et al. (2020), which examined firefighters who attended a fire during a 24-h shift, found naphthalene, phenanthrene, and anthracene (~34, 35, and 30 ng/g) to be highest. Baum et al. (2020) also found higher concentrations of LMW PAHs compared to HMW PAHs. Poutasse et al. (2020) used silicone dog tags worn in a necklace and placed over clothing, but underneath new turnout gear, for a period of 30 off-duty days and 30 on-duty days. Again, naphthalene and phenanthrene had the highest concentrations, i.e., 19.6 and 16.6 ng/g, respectively, for dog tags worn on duty. In a study conducted in North Carolina, which used SWBs worn by firefighters during six-day shifts, similar results were also found, i.e., phenanthrene having the highest median concentration (102.9, ng/g) for SWBs worn on shifts where the participant attended a fire (Levasseur et al. 2022). Surprisingly, in the Levasseur study, naphthalene had very low detection frequency, i.e., only detected in 15% of samples worn to a fire. Interestingly, in contrast to what was observed herein, fluoranthene and pyrene were the other two PAHs with highest SWB concentrations (Levasseur et al., 2020). Most recently, Bonner et al. (2023) reported PAH data from SWBs worn by mannequins in a staged fire. Wrist SWB concentrations were relatively similar but SWBs outside of PPE (i.e., on the jacket here or suspended in air in the Bonner study) were higher in the Bonner study, particularly for HMW PAHs where differences were ~100-fold. These differences were surprising considering the Bonner study exposed SWBs for an even shorter period than this study (i.e., <10 mins). It’s possible that the lack of movement of the suspended SWBs allowed for better sampling of HMW PAHs compared to the moving subjects of this study, and/or differences in fire conditions generated more HMW PAHs in the Bonner et al study. Overall, despite the fact that studies employed different designs and analytical methods, similar trends can be observed. Interestingly, no other study has found acenaphthylene to be in the top three highest concentrations compared to other PAHs. The differences in PAH results between the studies, particularly where firefighting took place, are likely due to variations in fire fuel and temperatures influencing PAH formation. Indeed, Banks et al. (2021) and Bakali et al. (2021) have noted markedly different PAH concentrations in fires of varying fuels.

The worker protection factor (WPF) had a significant, inverse correlation with molecular weight, suggesting that most of the deposition to the outer surfaces is happening in the vapour phase. Compared to previous studies, the WPF for total PAHs was within an order of magnitude of values previously reported by Wingfors et al. (2018) (i.e., 146), and those calculated using data from Kirk and Logan (2015) (i.e., 13), both of which were determined using active air sampler data. For individual PAHs, the WPFs calculated here were all lower than those reported by Wingfors et al. (2018) (i.e., 49 – 2200), but similar to data from Kirk & Logan (2015), i.e., 0.8 to 6.5-fold difference compared with this study. The most direct comparison can be made with Bonner et al. (2023) who analysed PAHs in SWBs on the wrist of mannequins and in the air next to the mannequins wearing two types of personal protective equipment (PPE). Similar patterns in WPFs were seen in that LMW PAHs had higher WPFs compared to HMW PAHs, likely due to the short deployment times that limited SWBs ability to accumulate HMW PAHs. However, the Bonner et al. study reported notably higher WPFs across all PAHs, possibly due to the apparent generally higher concentrations of environmental PAHs. Nonetheless, the results obtained here seem to indicate that firefighters' PPE can reduce LMW PAH exposure levels. However, it is evident that firefighters’ PPE does not fully protect them from PAH exposure as measurable amounts were still found on wrist SWBs. Improved design and fit of firefighter PPE, that, for example, closes the gap between the helmet and neck or jacket and glove, may further reduce exposures. The efficacy of such modifications, and/or exposure mitigation interventions, merit further investigation.

Higher sampling rates for LMW compared to HMW PAHs seen herein are similar to those previously reported in another passive sampler in the form of polyethylene semipermeable membrane devices (Čáslavský et al. 2004). Although generic gas-phase uptake rates for hung SWBs have been estimated to be $7.6 \pm 1.3\ \mathrm{m^3 d^{-1}\ dm^{-2}}$ at an air velocity of $1.3\ \mathrm{cm\ s^{-1}}$, sampling rates for SWBs worn as personal samplers have not been properly studied in a controlled environment (Hamzai et al. 2021). It has been suggested that SWB sampling rates in a personal sampler scenario have not been reported due to the amount of variability depending on the study (e.g., air velocity) (Tromp et al. 2019, Hamzai et al. 2021). Indeed, the results herein corroborate that notion, illustrating the effects the environment can have on SWBs’ ability to absorb PAHs.

Correlations between jacket and air PAHs illustrate that SWBs worn on jackets are in fact able to assess environmental levels of PAH; grouped PAHs (i.e., LMW, HMW, and total PAHs) from jacket SWBs correlated with nearly all ambient air PAHs levels. However, given limitations related to exposure duration, temperature, etc., it seems likely that the detected concentrations are comparatively low. The lack of correlations for individual HMW PAHs suggests that when using SWBs for short-term sampling, particularly in a fire environment, LMW PAH values are more reliable. Consequently, alternative sampling methods should be considered for HMW PAHs.

Regarding the use of SWBs for assessments of PAH exposure during firefighting, SWB deployment certainly constitutes a convenient approach compared with other personal air sampling methods, e.g., active personal air sampling. However, the influence of sampling duration time, temperature, and air turbulence on SWB PAH concentrations confounds the interpretation of the data obtained. Moreover, limitations in SWB sampling helps explain several of the patterns seen, including the low WPFs, $K_{SWB-Air}$, and sampling rates for HMW PAHs, lack of correlations between individual HMW PAHs on jacket SWBs and personal air samples, sporadic correlations between wrist and jacket SWB PAHs, and limited correlations between urinary PAHs metabolites with jacket and wrist SWB PAHs. HMW PAHs are particularly influenced, which is an important issue since HMW PAHs are most often known or probable human carcinogens (IARC 2010b). Further work is required to more appropriately understand the kinetics of SWB-PAHs interactions; moreover, to understand the effect environmental conditions (e.g., temperature, air velocity) on the ability to accurately measure PAH levels using SWBs in extreme environments (e.g., firefighting environments).

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References

Akomeah F, Nazir T, Martin GP, Brown MB. 2004. Effect of heat on the percutaneous absorption and skin retention of three model penetrants. Eur J Pharm Sci. 21:337–345, doi:10.1016/J.EJPS.2003.10.025.

Andersen MHG, Saber AT, Pedersen PB, Loft S, Hansen ÅM, Koponen IK, Pedersen JE, Ebbehøj N, Nørskov EC, Clausen PA, Garde AH, Vogel U, Møller P. 2017. Cardiovascular health effects following exposure of human volunteers during fire extinction exercises. Environ Heal A Glob Access Sci Source. 16:1–9, doi:10.1186/S12940-017-0303-8/TABLES/3.

Anderson KA, Points GL, Donald CE, Dixon HM, Scott RP, Wilson G, Tidwell LG, Hoffman PD, Herbstman JB, O'Connell SG. 2017. Preparation and performance features of wristband samplers and considerations for chemical exposure assessment. J Expo Sci Environ Epidemiol 2017 276. 27:551–559, doi:10.1038/jes.2017.9.

Arens E, Ghahramani A, Przybyla R, Andersen M, Min S, Peffer T, Raftery P, Zhu M, Luu V, Zhang H. 2020. Measuring 3D indoor air velocity via an inexpensive low-power ultrasonic anemometer. Energy Build. 211:109805, doi:10.1016/J.ENBUILD.2020.109805.

Bakali U, Baum JLR, Killawala C, Kobetz EN, Solle NS, Deo SK, Caban-Martinez AJ, Bachas LG, Daunert S. 2021. Mapping carcinogen exposure across urban fire incident response arenas using passive silicone-based samplers. Ecotoxicol Environ Saf. 228:112929, doi:10.1016/J.ECOENV.2021.112929.

Banks APW, Thai P, Engelsman M, Wang X, Osorio AF, Mueller JF. 2021. Characterising the exposure of Australian firefighters to polycyclic aromatic hydrocarbons generated in simulated compartment fires. Int J Hyg Environ Health. 231:113637, doi:10.1016/J.IJHEH.2020.113637.

Baum JLR, Bakali U, Killawala C, Santiago KM, Dikici E, Kobetz EN, Solle NS, Deo S, Bachas L, Daunert S. 2020. Evaluation of silicone-based wristbands as passive sampling systems using PAHs as an exposure proxy for carcinogen monitoring in firefighters: Evidence from the firefighter cancer initiative. Ecotoxicol Environ Saf. 205:111100, doi:10.1016/j.ecoenv.2020.111100.

Bonner EM, Horn GP, Smith DL, Kerber S, Fent KW, Tidwell LG, Scott RP, Adams KT, Anderson KA. 2023. Silicone passive sampling used to identify novel dermal chemical exposures of firefighters and assess PPE innovations. Int J Hyg Environ Health. 248:114095, doi:10.1016/J.IJHEH.2022.114095.

Caban-Martinez AJ, Louzado-Feliciano P, Santiago KM, Baum J, Schaefer Solle N, Rivera G, Miric M, Perez-Then E, Kobetz-Kerman EN, Daunert S. 2020. Objective Measurement of Carcinogens Among Dominican Republic Firefighters Using Silicone-Based Wristbands. J Occup Environ Med. 62:e611–e615.

Čáslavský J, Kotlaříková P, Benešová K. 2004. Sampling of airborne polycyclic aromatic hydrocarbons with semipermeable membrane devices. Environ Chem Lett. 2:89–92, doi:10.1007/S10311-004-0072-9/TABLES/1.

Chernyak YI, Shelepchikov AA, Brodsky ES, Grassman JA. 2012. PCDD, PCDF, and PCB exposure in current and former firefighters from Eastern Siberia. Toxicol Lett. 213:9–14, doi:10.1016/J.TOXLET.2011.09.021.

Daniels RD, Bertke S, Dahm MM, Yiin JH, Kubale TL, Hales TR, Baris D, Zahm SH, Beaumont JJ, Waters KM, Pinkerton LE. 2015. Exposure–response relationships for select cancer and non-cancer health outcomes in a cohort of US firefighters from San Francisco, Chicago and Philadelphia. Occup Env Med. 72:699–706.

Demers PA, DeMarini DM, Fent KW, Glass DC, Hansen J, Adetona O, Andersen MH, Freeman LEB, Caban-Martinez AJ, Daniels RD, Driscoll TR, Goodrich JM, Graber JM, Kirkham TL, Kjaerheim K, Kriebel D, Long AS, Main LC, Oliveira M, Peters S, Teras LR, Watkins ER, Burgess JL, Stec AA, White PA, DeBono NL, Benbrahim-Tallaa L, Conti A de, Ghissassi F El, et al. 2022. Carcinogenicity of occupational exposure as a firefighter. Lancet Oncol. 23:985–986, doi:10.1016/S1470-2045(22)00390-4.

Dixon HM, Scott RP, Holmes D, Calero L, Kincl LD, Waters KM, Camann DE, Calafat AM, Herbstman JB, Anderson KA. 2018. Silicone wristbands compared with traditional polycyclic aromatic hydrocarbon exposure assessment methods. Anal Bioanal Chem. 410:3059–3071, doi:10.1007/S00216-018-0992-Z/TABLES/4.

Donald CE, Scott RP, Wilson G, Hoffman PD, Anderson KA. 2019. Artificial turf: chemical flux and development of silicone wristband partitioning coefficients. Air Qual Atmos Heal. 12:597–611, doi:10.1007/S11869-019-00680-1/FIGURES/6.

Fent KW, Alexander B, Roberts J, Robertson S, Toennis C, Sammons D, Bertke S, Kerber S, Smith D, Horn G. 2017. Contamination of firefighter personal protective equipment and skin and the effectiveness of decontamination procedures. J Occup Environ Hyg. 14:801–814, doi:10.1080/15459624.2017.1334904.

Fent KW, Eisenberg J, Snawder J, Sammons D, Pleil JD, Stiegel MA, Mueller C, Horn GP, Dalton J. 2014. Systemic exposure to PAHs and benzene in firefighters suppressing controlled structure fires. Ann Occup Hyg. 58:830–845, doi:, https://doi.org/10.1093/annhyg/meu036.

Fernando S, Gallea M, VandenEnden L, House R, Verma D, Shaw L, McCarry BE, Shaw D, Britz-McKibbin P. 2016. Evaluation of Firefighter Exposure to Wood Smoke During Training Exercises at Burn Houses. Environ Sci Technol. 50:1536–1543, doi:https://doi.org/10.1021/acs.est.5b04752.

Hammel SC, Phillips AL, Hoffman K, Stapleton HM. 2018. Evaluating the Use of Silicone Wristbands to Measure Personal Exposure to Brominated Flame Retardants. Environ Sci Technol. 52:11875–11885, doi:10.1021/ACS.EST.8B03755/ASSET/IMAGES/LARGE/ES-2018-03755Q_0003.JPEG.

Hamzai L, Lopez Galvez N, Hoh E, Dodder NG, Matt GE, Quintana PJ. 2021. A systematic review of the use of silicone wristbands for environmental exposure assessment, with a focus on polycyclic aromatic hydrocarbons (PAHs). J Expo Sci Environ Epidemiol. 32:244–258, doi:10.1038/s41370-021-00359-9.

Harner T, Su K, Genualdi S, Karpowicz J, Ahrens L, Mihele C, Schuster J, Charland JP, Narayan J. 2013. Calibration and application of PUF disk passive air samplers for tracking polycyclic aromatic compounds (PACs). Atmos Environ. 75:123–128, doi:10.1016/J.ATMOSENV.2013.04.012.

Helsel D. 2009. Much ado about next to nothing: incorporating nondetects in science. Ann Occup Hyg. 54:257–262, doi:https://doi.org/10.1093/annhyg/mep092.

Horn GP, Kesler RM, Kerber S, Fent KW, Schroeder TJ, Scott WS, Fehling PC, Fernhall B, Smith DL. 2018. Thermal response to firefighting activities in residential structure fires: impact of job assignment and suppression tactic. Ergonomics. 61:404–419, doi:10.1080/00140139.2017.1355072.

IARC. 2010a. Painting, firefighting, and shiftwork. IARC Monogr Eval Carcinog Risks Hum. 98:9–764.

IARC. 2010b. Some non-heterocyclic polycyclic aromatic hydrocarbons and some related exposures. IARC Monogr Eval Carcinog Risks Hum. 92:1–853.

Keir J, Cakmak S, Blais J, White P. 2020a. The influence of demographic and lifestyle factors on urinary levels of PAH metabolites—empirical analyses of Cycle 2 (2009–2011) CHMS data. J Expo Sci Environ Epidemiol. 31:386–397, doi:10.1038/s41370-020-0208-4.

Keir JLA, Akhtar U, Matschke DMJ, Kirkham TL, Chan HM, Ayotte P, White PA, Blais JM. 2017. Elevated Exposures to Polycyclic Aromatic Hydrocarbons and Other Organic Mutagens in Ottawa Firefighters Participating in Emergency, On-Shift Fire Suppression. Environ Sci Technol. 51:12745–12755, doi:10.1021/acs.est.7b02850.

Keir JLA, Akhtar US, Matschke DMJ, White PA, Kirkham TL, Chan HM, Blais JM. 2020b. Polycyclic aromatic hydrocarbon (PAH) and metal contamination of air and surfaces exposed to combustion emissions during emergency fire suppression: Implications for firefighters' exposures. Sci Total Environ. 698:134211, doi:10.1016/j.scitotenv.2019.134211.

Keir JLA, Kirkham TL, Aranda-Rodriguez R, White PA, Blais JM. 2022. Effectiveness of dermal cleaning interventions for reducing firefighters' exposures to PAHs and genotoxins. Https://DoiOrg/101080/1545962420222150768. 1–16, doi:10.1080/15459624.2022.2150768.

Kerber S. 2010. Impact of Ventilation on Fire Behavior in Legacy and Contemporary Residential Construction. 1–405 p.

Kirk KM, Logan MB. 2015. Firefighting Instructors' Exposures to Polycyclic Aromatic Hydrocarbons During Live Fire Training Scenarios. J Occup Environ Hyg. 12:227–234, doi:10.1080/15459624.2014.955184.

Levasseur JL, Hoffman K, Herkert NJ, Cooper E, Hay D, Stapleton HM. 2022. Characterizing firefighter's exposure to over 130 SVOCs using silicone wristbands: A pilot study comparing on-duty and off-duty exposures. Sci Total Environ. 834:155237, doi:10.1016/J.SCITOTENV.2022.155237.

Mayer AC, Horn GP, Fent KW, Bertke SJ, Kerber S, Kesler RM, Newman H, Smith DL. 2020. Impact of select PPE design elements and repeated laundering in firefighter protection from smoke exposure. J Occup Environ Hyg. 17:505–514, doi:10.1080/15459624.2020.1811869/SUPPL_FILE/UOEH_A_1811869_SM1394.DOCX.

Mendoza-Sanchez I, Uwak I, Myatt L, Cleve A Van, Pulczinski JC, Rychlik KA, Sweet S, Ramani T, Zietsman J, Zamora ML, Koehler K, Carrillo G, Johnson NM. 2021. Maternal exposure to polycyclic aromatic hydrocarbons in South Texas, evaluation of silicone wristbands as personal passive samplers. J Expo Sci Environ Epidemiol 2021 322. 32:280–288, doi:10.1038/s41370-021-00348-y.

O'Connell SG, Anderson KA, Epstein MI. 2021. Determining chemical air equivalency using silicone personal monitors. J Expo Sci Environ Epidemiol 2021 322. 32:268–279, doi:10.1038/s41370-021-00332-6.

O'Connell SG, Kincl LD, Anderson KA. 2014. Silicone wristbands as personal passive samplers. Environ Sci Technol. 48:3327–3335, doi:10.1021/es405022f.

Park JS, Voss RW, McNeel S, Wu N, Guo T, Wang Y, Israel L, Das R, Petreas M. 2015. High exposure of California firefighters to polybrominated diphenyl ethers. Environ Sci Technol. 49:2948–2958, doi:10.1021/es5055918.

Pinkerton L, Bertke SJ, Yiin J, Dahm M, Kubale T, Hales T, Purdue M, Beaumont JJ, Daniels R. 2020. Mortality in a cohort of US firefighters from San Francisco, Chicago and Philadelphia: An update. Occup Environ Med. 77:84–93, doi:10.1136/oemed-2019-105962.

Pleil JD, Stiegel MA, Fent KW. 2014. Exploratory breath analyses for assessing toxic dermal exposures of firefighters during suppression of structural burns. J Breath Res. 8:37107.

Poutasse CM, Poston WSC, Jahnke SA, Haddock CK, Tidwell LG, Hoffman PD, Anderson KA. 2020. Discovery of firefighter chemical exposures using military-style silicone dog tags. Environ Int. 142:105818, doi:10.1016/j.envint.2020.105818.

Pozo K, Harner T, Lee SC, Wania F, Muir DCG, Jones KC. 2009. Seasonally resolved concentrations of persistent organic pollutants in the global atmosphere from the first year of the GAPS Study. Environ Sci Technol. 43:796–803, doi:10.1021/ES802106A/SUPPL_FILE/ES802106A_SI_003.PDF.

Randall G. 2020. Characterization of a Live Fire Training Simulator for use in the Canadian Fire Service.

Santos PM, Nogal Sánchez M del, Pérez Pavón JL, Cordero BM, Fernández RV. 2019. Liquid-liquid extraction-programmed temperature vaporizer-gas chromatography-mass spectrometry for the determination of polycyclic aromatic hydrocarbons in saliva samples. Application to the occupational exposure of firefighters. Talanta. 192:69–78, doi:10.1016/J.TALANTA.2018.09.030.

Sjöström M, Julander A, Strandberg B, Lewné M, Bigert C. 2019. Airborne and Dermal Exposure to Polycyclic Aromatic Hydrocarbons, Volatile Organic Compounds, and Particles among Firefighters and Police Investigators. Ann Work Expo Heal. 63:533–545, doi:10.1093/annweh/wxz030.

Sritharan J, Kirkham TL, MacLeod J, Marjerrison N, Lau A, Dakouo M, Logar-Henderson C, Norzin T, DeBono NL, Demers PA. 2022. Cancer risk among firefighters and police in the Ontario workforce. Occup Environ Med. 79:533–539, doi:10.1136/OEMED-2021-108146.

Tromp PC, Beeltje H, Okeme JO, Vermeulen R, Pronk A, Diamond ML. 2019. Calibration of polydimethylsiloxane and polyurethane foam passive air samplers for measuring semi volatile organic compounds using a novel exposure chamber design. Chemosphere. 227:435–443, doi:10.1016/J.CHEMOSPHERE.2019.04.043.

Trowbridge J, Gerona RR, Lin T, Rudel RA, Bessonneau V, Buren H, Morello-Frosch R. 2020. Exposure to Perfluoroalkyl Substances in a Cohort of Women Firefighters and Office Workers in San Francisco. Environ Sci Technol. 54:3363–3374, doi:10.1021/acs.est.9b05490.

Wingfors H, Nyholm JR, Magnusson R, Wijkmark CH. 2018. Impact of Fire Suit Ensembles on Firefighter PAH Exposures as Assessed by Skin Deposition and Urinary Biomarkers. Ann Work Expo Heal. 62:221–231, doi:10.1093/annweh/wxx097.

Young AS, Herkert N, Stapleton HM, Cedeño Laurent JG, Jones ER, MacNaughton P, Coull BA, James-Todd T, Hauser R, Luna ML, Chung YS, Allen JG. 2021. Chemical contaminant exposures assessed using silicone wristbands among occupants in office buildings in the USA, UK, China, and India. Environ Int. 156:106727, doi:10.1016/J.ENVINT.2021.106727.

Supplementary Information

Chemical analyses

The PAH native standard mix used consisted of 16 PAHs: naphthalene, acenaphthylene, acenaphthene, fluorene, phenanthrene, anthracene, fluoranthene, pyrene, benzo[*a*]anthracene, chrysene, benzo[*b*]fluoranthene, benzo[*k*]fluoranthene, benzo[*a*]pyrene, indeno[1,2,3-*c,d*]pyrene, dibenzo[*a,h*]anthracene, benzo[*g,h,i*]perylene.

The PAH labelled internal standard mix used consisted of 16 deuterated PAHs: naphthalene-d8, acenaphthylene-d8, acenaphthene-d10, fluorene-d10, phenanthrene-d10, anthracene-d10, fluoranthene-d10, pyrene-d10, benzo[*a*]anthracene-d12, chrysene-d12, benzo[*b*]fluoranthene-d12, benzo[*k*]fluoranthene-d12, benzo[*a*]pyrene-d12, indeno [1,2,3-*c,d*]pyrene-d12, dibenzo[*a,h*]anthracene-d14, benzo[*g,h,i*]perylene-d12).

The analytical instrument used was a TSQ Quantum XLS Ultra™ Gas Chromatography-triple quadrupole mass spectrometer (GC-MS/MS) (ThermoFisher Scientific, MA). Trace GC Ultra supplied with a split/splitless injector and operated for hot splitless injection of 0.8 min splitless mode, a split flow of 30 mL/min. The GC was fitted with a DB-EUPAH GC column (30 m x 0.25 mm I.D x 0.25 µm film; Phenomenex, CA).The analytical column was maintained at an initial oven temperature of 55°C held for 2 min, then ramped at 18°C/min to 320°C held for 1 minute at final temperature. The carrier gas was helium and was maintained at a constant flow rate of 1.0 mL/min. Analytes were eluted to the TSQ-8000 mass spectrometer equipped with an electron ionization source at 50 µA in selected reaction monitoring (SRM) mode. The source temperature was 300°C and transfer line temperature was 280°C. The MS contained a triple quadrupole mass analyzer, with Q1 and Q3 peak width set at 0.7 (FWHM) and a cycle time of 0.2 seconds. Details of the run can be found in Table A1.

Table S3-1. Details of analytical run.

Name	RT	Precursor Mass	Product Mass	Collision Energy
naphthalene-d8	5.98	136.1	108.1	20
naphthalene	6.004	128.1	102	15
acenaphthylene-d8	7.752	160.1	158.1	15
acenaphthylene	7.769	152.1	126.1	25
acenaphthylene	7.769	152.1	151.1	15
acenaphthene-d10	7.854	164.2	162.2	15
acenaphthene	7.887	153.1	127.1	25
acenaphthene	7.895	153.1	152.1	15
fluorene-d10	8.361	176.2	174.2	20
fluorene	8.398	166.1	165.1	15
phenanthrene-d10	9.514	188.2	160.1	20
phenanthrene	9.545	178.1	152.1	20
anthracene-d10	9.548	188.2	160.1	20
anthracene	9.572	178.1	152.1	20
fluoranthene-d10	10.902	212.2	208.1	35
fluoranthene	10.932	202.1	200.1	30
p-terphenyl-d14	11.242	244.2	240.2	25
pyrene-d10	11.32	212.2	208.2	35
pyrene	11.351	202.1	200.1	35
benz[*a*]anthracene-d12	13.368	240.2	236.2	30
benz[*a*]anthracene	13.426	228.1	226.2	30
chrysene-d12	13.548	240.2	236.1	30
chrysene	13.62	228.1	226.1	30
benzo[*b*]fluoranthene-d12	16.15	264.2	260.2	30
benzo[*k*]fluoranthene-d12	16.228	264.2	260.1	35
benzo[*b*]fluoranthene	16.232	252.1	250.1	30
benzo[*k*]fluoranthene	16.3	252.1	250.1	30
benzo[*a*]pyrene-d12	17.395	264.2	260.2	40
benzo[*a*]pyrene	17.48	252.1	250.1	35
dibenzo[*a*,*h*]anthracene-d14	20.783	292.2	288.2	35
indeno[1,2,3-*cd*]pyrene-d12	20.851	288.2	284.2	40
dibenz[*a*,*h*]anthracene	20.922	278.1	276.1	30
indeno[1,2,3-*cd*]pyrene	20.956	276.1	274.1	35
benzo[*g*,*h*,*i*]perylene-d12	22.208	288.2	284.2	45
benzo[*g*,*h*,*i*]perylene	22.327	276.1	274.1	40

Table S3-2. Comparison of WPFs between Keir et al wrist SWB training fire data with wrist SWB data from Bonner et al (2023).

	Keir et al. data	Bonner et al. (2023)	
PAH	**Wrist WPF**	**Wrist WPF – Standard PPE**	**Wrist WPF - One-piece PPE**
Naphthalene	41.6	638.0	572.0
Acenaphthylene	213.8	1980.0	1730.0
Acenaphthene	79.4	190.0	256.0
Fluorene	70.3	1840.0	2370.0
Phenanthrene	68.3	547.0	1010.0
Anthracene	123.8	1570.0	1570.0
Fluoranthene	16.8	162.0	2490.0
Pyrene	25.0	127.0	2030.0
Benzo[*a*]anthracene	NA	61.5	510.0
Chrysene	NA	251.0	714.0
Benzo[*b*]fluoranthene	6.6	57.4	1100.0
Benzo[*k*]fluoranthene	NA	33.9	288.0
Benzo[*a*]pyrene	5.5	45.9	334.0
Indeno[1,2,3-cd]pyrene	NA	35.2	945.0
Dibenzo[*a*,h]anthracene	NA	NA	NA
Benzo[*g*,*h*,*i*]perylene	2.8	40.8	691.0
Average LMW PAHs	87.7	989.6	1428.3
SD LMW PAHs	64.7	783.7	857.1
Average HMW PAHs	10.0	81.6	826.5
SD HMW PAHs	9.9	74.8	560.7

NA indicates >80% below detection for Keir et al., missing value for Bonner et al.

Table S3-3. Sampling rates (R) in m^3 day^{-1} for jacket SWBs (n=14). Some SWBs did not have matching air data and were omitted from sampling rate calculations.

	Sampling Rate, R (m^3 day^{-1})			
PAH	GM	St Dev	Min	Max
Naphthalene	0.29	0.36	0.07	1.28
Acenaphthylene	0.32	0.19	0.07	0.80
Acenaphthene	0.64	0.40	0.18	1.53
Fluorene	0.54	0.36	0.10	1.27
Phenanthrene	0.34	0.34	0.08	1.18
Anthracene	0.34	0.28	0.07	0.88
Fluoranthene	0.16	0.18	0.04	0.55
Pyrene	0.12	0.14	0.03	0.43
Benzo[*a*]anthracene	0.04	0.09	0.01	0.30
Chrysene	0.04	0.09	0.01	0.28
Benzo[*b*]fluoranthene	0.04	0.10	0.01	0.32
Benzo[*k*]fluoranthene	0.01	0.05	0.003	0.17
Benzo[*a*]pyrene	0.03	0.09	0.004	0.33
Indeno[1,2,3-cd]pyrene	0.02	0.14	0.003	0.52
Benzo[*g*,*h*,*i*]perylene	0.03	0.26	0.004	0.91
Total LMW PAHs	0.29	0.30	0.05	0.98
Total HMW PAHs	0.15	0.29	0.01	0.92
Total PAHs	0.28	0.30	0.05	0.97

GM= geometric mean; St Dev = standard deviation, Min= minimum, Max= maximum.

Table S3-4. Abbreviations for Tables S3-4 to S3-6.

Abbreviation	**PAH**
Nap	Naphthalene
Acey	Acenaphthylene
Ace	Acenaphthene
Fluo	Fluorene
Dibenzoth	Dibenzothiophene
Phen	Phenanthrene
Anth	Anthracene
Fluoranth	Fluoranthene
Pyr	Pyrene
Ret	Retene
Benzoa	Benzo[*a*]anthracene
Chyrs	Chrysene
Benzobfl	Benzo[*b*]fluoranthene
Benzokfl	Benzo[*k*]fluoranthene
BaP	Benzo[*a*]pyrene
BeP	Benzo[*e*]pyrene
Indeno	Indeno[1,2,3-*cd*]pyrene
Dibenzoah	Dibenzo[*a*,*h*]anthracene
Benzoghi	Benzo[*g*,*h*,*i*]perylene
LMW	Total LMW PAHs
HMW	Total HMW PAHs
Total	Total PAHs
Post-fire OHP	Hydroxypyrene
Post-fire ΣOH-Fluo	Sum of fluorene metabolites (i.e., 2- 3-, and 9-hydroxyfluorene)
Post-fire ΣOH-Nap	Sum of naphthalene metabolites (i.e., 1- and 2-hydroxynaphthalene)
Post-fire ΣOH-Phen	Sum of phenanthrene metabolites (i.e., 1-, 2-, 3-. 4-, and 9-hydroxyphenanthrene)
Post-fire total	Sum of all PAH metabolites
Fold change	Ratio between urinary PAH metabolites concentrations after the fire (i.e., post-fire) compared to before (i.e., pre-fire)

Table S3-5. Pearson correlation coefficients (top number) and *p* values (bottom number) of PAHs from silicone wristbands (SWBs) worn on the jacket versus wrist (N=20). All data were log transformed prior to analyses. Values are bolded where *p*<0.05.

	Jacket SWB																	
Wrist SWB	Nap	Acey	Ace	Fluo	Phen	Anth	Fluoranth	Pyr	Benzoa	Chyrs	Benzobfl	Benzokfl	BaP	Indeno	Benzoghi	LMW	HMW	Total
Nap	0.166	0.127	0.177	0.067	0.159	0.101	0.182	0.151	0.199	0.186	0.211	-0.034	0.207	0.168	0.200	0.157	0.164	0.160
	0.485	0.594	0.454	0.778	0.504	0.672	0.442	0.526	0.401	0.433	0.371	0.886	0.382	0.478	0.399	0.508	0.491	0.500
Acey	0.064	-0.002	0.123	-0.017	0.089	0.071	0.127	0.088	0.035	0.035	0.032	-0.250	0.086	0.084	0.094	0.057	0.077	0.060
	0.789	0.992	0.606	0.943	0.710	0.766	0.595	0.712	0.884	0.883	0.894	0.287	0.719	0.726	0.692	0.812	0.747	0.802
Ace	0.168	0.117	0.204	0.187	0.148	0.220	0.291	0.275	0.143	0.131	0.036	-0.229	0.075	0.067	0.061	0.168	0.222	0.172
	0.478	0.624	0.387	0.430	0.534	0.352	0.213	0.240	0.546	0.583	0.880	0.332	0.753	0.778	0.799	0.479	0.347	0.467
Fluo	0.214	0.127	0.243	0.132	0.233	0.226	0.296	0.255	0.177	0.166	0.120	-0.092	0.172	0.194	0.252	0.206	0.254	0.212
	0.364	0.595	0.302	0.580	0.323	0.338	0.206	0.277	0.455	0.483	0.613	0.699	0.468	0.412	0.285	0.383	0.279	0.371
Phen	0.142	0.000	0.053	0.011	0.172	0.221	0.313	0.281	0.326	0.319	0.284	0.103	0.295	0.334	0.449	0.118	0.319	0.129
	0.549	0.999	0.825	0.964	0.470	0.348	0.179	0.230	0.161	0.170	0.226	0.665	0.206	0.150	**0.047**	0.620	0.170	0.587
Anth	0.318	0.189	0.357	0.272	0.342	0.402	0.504	0.475	0.343	0.315	0.229	-0.036	0.231	0.244	0.290	0.310	0.449	0.319
	0.171	0.425	0.123	0.247	0.141	0.079	**0.024**	**0.035**	0.138	0.176	0.332	0.880	0.327	0.300	0.216	0.184	**0.047**	0.170
Fluoranth	0.372	0.220	0.208	0.158	0.320	0.364	0.468	0.434	0.575	0.560	0.467	0.373	0.528	0.552	0.720	0.333	0.493	0.343
	0.107	0.351	0.380	0.505	0.170	0.114	**0.037**	0.056	**0.008**	**0.010**	**0.038**	0.106	**0.017**	**0.012**	**0.000**	0.152	**0.027**	0.139
Pyr	0.375	0.191	0.220	0.180	0.345	0.377	0.511	0.477	0.596	0.573	0.482	0.464	0.529	0.560	0.730	0.333	0.541	0.344
	0.103	0.421	0.351	0.449	0.136	0.101	**0.021**	**0.034**	**0.006**	**0.008**	**0.031**	**0.039**	**0.017**	**0.010**	**0.000**	0.152	**0.014**	0.137
Benzobfl	-0.457	-0.517	-0.492	-0.275	-0.260	-0.229	-0.281	-0.242	-0.176	-0.152	0.080	-0.321	-0.314	-0.270	-0.237	-0.442	-0.249	-0.436
	0.043	**0.020**	**0.028**	0.241	0.269	0.331	0.230	0.304	0.458	0.524	0.738	0.167	0.178	0.249	0.315	0.051	0.290	0.055
BaP	-0.484	-0.427	-0.258	-0.205	-0.309	-0.276	-0.303	-0.233	-0.372	-0.352	-0.156	-0.276	-0.427	-0.451	-0.586	-0.442	-0.287	-0.438
	0.031	0.061	0.271	0.386	0.185	0.240	0.194	0.322	0.107	0.128	0.512	0.240	0.060	**0.046**	**0.007**	0.051	0.220	0.054
Dibenzoah	0.388	0.357	0.365	0.215	0.332	0.440	0.498	0.493	0.535	0.502	0.399	0.458	0.541	0.544	0.531	0.376	0.547	0.387
	0.091	0.122	0.114	0.363	0.152	0.053	**0.026**	**0.027**	**0.015**	**0.024**	0.081	**0.042**	**0.014**	**0.013**	**0.016**	0.103	**0.013**	0.092
Benzoghi	0.472	0.371	0.335	0.309	0.483	0.487	0.574	0.558	0.738	0.728	0.682	0.730	0.737	0.751	0.897	0.454	0.657	0.468

	0.035	0.107	0.149	0.186	**0.031**	**0.029**	**0.008**	**0.011**	**0.000**	**0.000**	**0.001**	**0.000**	**0.000**	**0.000**	**<.0001**	**0.044**	**0.002**	**0.037**
LMW	0.191	0.138	0.204	0.088	0.186	0.140	0.220	0.186	0.212	0.198	0.205	-0.044	0.214	0.186	0.223	0.182	0.196	0.185
	0.419	0.561	0.388	0.711	0.431	0.556	0.350	0.434	0.369	0.403	0.386	0.853	0.364	0.432	0.344	0.443	0.407	0.434
HMW	0.074	-0.081	-0.012	-0.002	0.175	0.242	0.336	0.338	0.510	0.506	0.579	0.314	0.451	0.483	0.602	0.057	0.402	0.075
	0.755	0.735	0.959	0.993	0.461	0.304	0.147	0.145	**0.022**	**0.023**	**0.008**	0.177	**0.046**	**0.031**	**0.005**	0.811	0.079	0.755
Total	0.171	0.106	0.188	0.073	0.180	0.138	0.224	0.191	0.226	0.214	0.240	-0.041	0.230	0.202	0.242	0.161	0.204	0.166
	0.471	0.657	0.428	0.759	0.447	0.561	0.344	0.420	0.339	0.366	0.308	0.865	0.330	0.392	0.305	0.498	0.388	0.486

Table S3-6. Pearson correlation coefficients (top number) and *p* values (bottom number) of PAHs from silicone wristbands (SWBs) worn on the jacket versus personal air concentrations. N=14 since not all participants who wore wristbands also wore a personal air sampler. All data were log transformed prior to analyses. Values are bolded where $p<0.05$.

	Personal Air																					
Jacket SWB	Nap	Acey	Ace	Fluo	Dibenzoth	Phen	Anth	Fluoranth	Pyr	Retene	Benzoa	Chyrs	Benzobfl	Benzokfl	BeP	BaP	Indeno	Dibenzah	Benzoghi	LMW	HMW	Total
Nap	0.34	0.70	0.74	0.74	0.49	0.71	0.69	0.78	0.74	0.82	0.75	0.76	0.77	0.73	0.74	0.73	0.76	0.73	0.77	0.59	0.76	0.56
	0.236	**0.005**	**0.002**	**0.002**	0.076	**0.004**	**0.007**	**0.001**	**0.003**	**0.000**	**0.002**	**0.002**	**0.001**	**0.003**	**0.003**	**0.003**	**0.002**	**0.003**	**0.001**	**0.027**	**0.001**	**0.039**
Acey	0.37	0.69	0.72	0.73	0.48	0.72	0.65	0.77	0.72	0.76	0.74	0.74	0.75	0.74	0.73	0.73	0.77	0.72	0.72	0.60	0.75	0.57
	0.187	**0.006**	**0.003**	**0.003**	0.084	**0.004**	**0.011**	**0.001**	**0.004**	**0.002**	**0.002**	**0.002**	**0.002**	**0.003**	**0.003**	**0.003**	**0.001**	**0.004**	**0.004**	**0.023**	**0.002**	**0.032**
Ace	0.45	0.73	0.78	0.77	0.47	0.80	0.73	0.80	0.75	0.74	0.78	0.79	0.80	0.77	0.78	0.77	0.77	0.78	0.80	0.70	0.78	0.68
	0.106	**0.003**	**0.001**	**0.001**	0.092	**0.001**	**0.003**	**0.001**	**0.002**	**0.003**	**0.001**	**0.001**	**0.001**	**0.001**	**0.001**	**0.001**	**0.001**	**0.001**	**0.001**	**0.005**	**0.001**	**0.007**
Fluo	0.47	0.56	0.62	0.59	0.54	0.62	0.58	0.66	0.59	0.62	0.61	0.63	0.65	0.61	0.63	0.61	0.62	0.61	0.69	0.58	0.63	0.57
	0.089	**0.035**	**0.018**	**0.026**	**0.048**	**0.017**	**0.031**	**0.011**	**0.026**	**0.018**	**0.019**	**0.017**	**0.013**	**0.020**	**0.015**	**0.022**	**0.018**	**0.020**	**0.006**	**0.029**	**0.016**	**0.034**
Phen	0.35	0.55	0.61	0.59	0.43	0.58	0.56	0.64	0.58	0.67	0.59	0.61	0.63	0.58	0.59	0.57	0.61	0.58	0.67	0.48	0.61	0.46
	0.221	**0.041**	**0.021**	**0.026**	0.127	**0.030**	**0.037**	**0.013**	**0.031**	**0.008**	**0.027**	**0.021**	**0.016**	**0.031**	**0.026**	**0.035**	**0.021**	**0.028**	**0.009**	0.083	**0.020**	0.100
Anth	0.25	0.56	0.61	0.60	0.52	0.58	0.58	0.63	0.57	0.66	0.58	0.61	0.62	0.57	0.58	0.56	0.59	0.59	0.65	0.44	0.61	0.41
	0.391	**0.036**	**0.020**	**0.024**	0.058	**0.030**	**0.029**	**0.016**	**0.032**	**0.010**	**0.028**	**0.022**	**0.019**	**0.033**	**0.029**	**0.036**	**0.025**	**0.025**	**0.013**	0.119	**0.022**	0.146
Fluoranth	0.15	0.61	0.66	0.65	0.48	0.62	0.62	0.67	0.61	0.71	0.61	0.63	0.65	0.59	0.61	0.59	0.62	0.61	0.69	0.41	0.64	0.37
	0.616	**0.019**	**0.010**	**0.012**	0.084	**0.019**	**0.017**	**0.009**	**0.021**	**0.005**	**0.021**	**0.015**	**0.012**	**0.026**	**0.021**	**0.028**	**0.019**	**0.020**	**0.006**	0.146	**0.014**	0.187
Pyr	0.14	0.60	0.65	0.63	0.50	0.61	0.62	0.65	0.59	0.68	0.59	0.61	0.63	0.57	0.59	0.57	0.59	0.60	0.68	0.40	0.62	0.37
	0.629	**0.023**	**0.012**	**0.016**	0.069	**0.021**	**0.019**	**0.012**	**0.027**	**0.008**	**0.027**	**0.020**	**0.016**	**0.032**	**0.026**	**0.034**	**0.025**	**0.023**	**0.007**	0.155	**0.018**	0.196
Benzoa	0.06	0.42	0.46	0.48	0.24	0.38	0.45	0.49	0.43	0.61	0.42	0.45	0.44	0.40	0.39	0.38	0.44	0.40	0.45	0.22	0.46	0.19
	0.827	0.133	0.095	0.084	0.400	0.178	0.108	0.075	0.124	**0.021**	0.137	0.108	0.114	0.158	0.163	0.185	0.117	0.155	0.105	0.457	0.101	0.524
Chrys	0.05	0.35	0.39	0.40	0.18	0.31	0.38	0.42	0.35	0.53	0.34	0.37	0.37	0.32	0.32	0.30	0.37	0.33	0.38	0.15	0.38	0.12
	0.874	0.226	0.173	0.154	0.538	0.283	0.184	0.138	0.217	**0.050**	0.231	0.192	0.199	0.258	0.267	0.295	0.199	0.253	0.180	0.616	0.180	0.684

Benzobfl	0.05	0.16	0.21	0.20	0.19	0.15	0.22	0.24	0.15	0.31	0.15	0.18	0.19	0.14	0.15	0.12	0.17	0.14	0.27	0.04	0.19	0.03
	0.865	0.584	0.472	0.487	0.524	0.601	0.440	0.404	0.599	0.276	0.605	0.532	0.517	0.642	0.614	0.694	0.560	0.635	0.358	0.885	0.509	0.923
Benzokfl	-0.03	0.36	0.40	0.44	0.14	0.34	0.40	0.45	0.39	0.57	0.38	0.41	0.40	0.33	0.33	0.33	0.38	0.30	0.41	0.14	0.41	0.10
	0.920	0.211	0.154	0.117	0.639	0.239	0.155	0.110	0.171	**0.033**	0.184	0.147	0.158	0.242	0.243	0.255	0.183	0.290	0.146	0.640	0.144	0.725
BaP	-0.02	0.29	0.33	0.38	0.09	0.24	0.33	0.37	0.32	0.56	0.31	0.35	0.33	0.26	0.25	0.25	0.33	0.25	0.33	0.08	0.34	0.05
	0.948	0.312	0.247	0.183	0.752	0.404	0.245	0.188	0.260	**0.037**	0.276	0.227	0.249	0.364	0.390	0.390	0.246	0.390	0.248	0.791	0.227	0.869
Indeno	-0.01	0.25	0.29	0.34	0.03	0.19	0.29	0.33	0.30	0.56	0.28	0.31	0.30	0.22	0.21	0.21	0.30	0.21	0.29	0.05	0.31	0.03
	0.964	0.384	0.317	0.235	0.907	0.525	0.321	0.251	0.303	**0.038**	0.338	0.278	0.303	0.444	0.471	0.467	0.304	0.469	0.312	0.855	0.280	0.928
Benzoghi	-0.01	0.28	0.31	0.36	0.04	0.20	0.31	0.35	0.32	0.58	0.30	0.33	0.32	0.24	0.23	0.23	0.31	0.23	0.32	0.07	0.33	0.04
	0.967	0.340	0.282	0.207	0.883	0.483	0.282	0.221	0.267	**0.031**	0.301	0.245	0.265	0.403	0.425	0.422	0.276	0.424	0.267	0.819	0.247	0.894
LMW	0.36	0.68	0.73	0.73	0.49	0.71	0.67	0.76	0.72	0.79	0.73	0.75	0.75	0.72	0.73	0.71	0.75	0.72	0.76	0.59	0.75	0.56
	0.203	**0.007**	**0.003**	**0.003**	0.074	**0.005**	**0.009**	**0.002**	**0.004**	**0.001**	**0.003**	**0.002**	**0.002**	**0.004**	**0.003**	**0.004**	**0.002**	**0.004**	**0.002**	**0.027**	**0.002**	**0.038**
HMW	0.12	0.57	0.62	0.61	0.45	0.56	0.58	0.62	0.56	0.68	0.56	0.59	0.60	0.54	0.56	0.54	0.57	0.56	0.65	0.36	0.59	0.33
	0.671	**0.034**	**0.019**	**0.022**	0.109	**0.036**	**0.029**	**0.017**	**0.036**	**0.007**	**0.037**	**0.027**	**0.023**	**0.045**	**0.039**	**0.049**	**0.032**	**0.036**	**0.013**	0.207	**0.025**	0.256
Total	0.35	0.68	0.73	0.72	0.49	0.70	0.67	0.76	0.71	0.79	0.73	0.74	0.75	0.72	0.72	0.71	0.75	0.71	0.75	0.58	0.74	0.55
	0.215	**0.007**	**0.003**	**0.003**	0.075	**0.005**	**0.009**	**0.002**	**0.004**	**0.001**	**0.003**	**0.002**	**0.002**	**0.004**	**0.004**	**0.005**	**0.002**	**0.004**	**0.002**	**0.030**	**0.002**	**0.042**

Table S3-7. Pearson correlation coefficients (top number) and *p* values (middle number) of PAHs from silicone wristbands (SWBs) worn on the jacket versus urinary PAH metabolite metrics. N=bottom number; not all participants who wore wristbands had measurable amounts of urinary PAH metabolites. Fold-change is the ratio of PAH metabolites post-fire compared to pre-fire. All data were log transformed prior to analyses. Values are bolded where $p<0.05$.

		Jacket SWB																	
		Nap	Acey	Ace	Fluo	Phen	Anth	Fluoranth	Pyr	Benzoa	Chrys	Benzobfl	Benzokfl	BaP	Indeno	Benzoghi	LMW	HMW	Total
Urinary PAH Metabolites	Post-fire OHP	-0.04	0.01	-0.32	-0.11	0.02	-0.08	-0.13	-0.15	0.06	0.08	0.08	0.01	-0.04	-0.03	0.06	-0.04	-0.12	-0.04
		0.86	0.98	0.16	0.64	0.93	0.73	0.58	0.52	0.81	0.73	0.72	0.96	0.87	0.89	0.79	0.87	0.60	0.86
		20	20	20	20	20	20	20	20	20	20	20	20	20	20	20	20	20	20
	Post-fire ΣOH-Fluo	0.04	0.04	-0.17	-0.01	0.10	-0.04	-0.09	-0.13	-0.02	-0.03	-0.03	0.01	-0.11	-0.07	0.04	0.04	-0.11	0.03
		0.88	0.86	0.48	0.97	0.69	0.86	0.70	0.58	0.92	0.92	0.89	0.97	0.66	0.76	0.87	0.88	0.65	0.91
		20	20	20	20	20	20	20	20	20	20	20	20	20	20	20	20	20	20
	Post-fire ΣOH-Nap	0.39	0.34	0.23	0.42	0.40	0.33	0.35	0.37	0.46	0.51	0.40	0.51	0.32	0.26	0.42	0.39	0.39	0.39
		0.11	0.17	0.35	0.08	0.10	0.18	0.15	0.13	0.05	**0.03**	0.10	**0.03**	0.19	0.31	0.08	0.11	0.11	0.11
		18	18	18	18	18	18	18	18	18	18	18	18	18	18	18	18	18	18
	Post-fire ΣOH-Phen	0.15	0.19	0.06	0.17	0.27	0.15	0.10	0.07	0.13	0.13	0.10	0.08	-0.03	-0.05	0.04	0.17	0.07	0.17
		0.56	0.46	0.82	0.50	0.28	0.54	0.69	0.77	0.61	0.62	0.68	0.76	0.92	0.85	0.86	0.50	0.79	0.51
		18	18	18	18	18	18	18	18	18	18	18	18	18	18	18	18	18	18
	Post-fire total	0.42	0.38	0.27	0.44	0.46	0.40	0.43	0.45	0.54	0.59	0.47	0.51	0.38	0.30	0.48	0.42	0.46	0.43
		0.09	0.14	0.29	0.08	0.07	0.11	0.08	0.07	**0.03**	**0.01**	0.05	**0.04**	0.13	0.24	0.05	0.09	0.06	0.09
		17	17	17	17	17	17	17	17	17	17	17	17	17	17	17	17	17	17
	Fold change OHP	0.25	0.30	0.14	0.27	0.41	0.39	0.29	0.27	0.31	0.33	0.18	0.21	0.16	0.15	0.21	0.28	0.28	0.28
		0.31	0.21	0.57	0.26	0.08	0.10	0.22	0.27	0.20	0.17	0.47	0.40	0.51	0.53	0.38	0.24	0.24	0.24
		19	19	19	19	19	19	19	19	19	19	19	19	19	19	19	19	19	19
	Fold change ΣOH-Fluo	0.20	0.12	0.02	0.21	0.28	0.29	0.19	0.17	0.35	0.34	0.21	0.09	0.26	0.31	0.29	0.20	0.22	0.20
		0.40	0.61	0.95	0.38	0.23	0.21	0.41	0.47	0.13	0.14	0.38	0.70	0.27	0.19	0.22	0.41	0.36	0.40
		20	20	20	20	20	20	20	20	20	20	20	20	20	20	20	20	20	20
	Fold change ΣOH-Nap	0.15	0.18	0.07	0.13	0.13	0.10	0.07	0.06	0.19	0.15	0.15	0.20	0.26	0.22	0.09	0.15	0.10	0.15
		0.54	0.48	0.79	0.59	0.61	0.68	0.78	0.80	0.44	0.54	0.56	0.42	0.30	0.39	0.73	0.55	0.69	0.56
		18	18	18	18	18	18	18	18	18	18	18	18	18	18	18	18	18	18
	Fold change ΣOH-Phen	0.35	0.43	0.34	0.40	0.45	0.44	0.29	0.25	0.23	0.24	-0.02	0.09	0.08	0.08	0.05	0.39	0.24	0.39
		0.18	0.10	0.20	0.13	0.08	0.09	0.28	0.34	0.39	0.37	0.95	0.74	0.76	0.77	0.86	0.14	0.36	0.14
		16	16	16	16	16	16	16	16	16	16	16	16	16	16	16	16	16	16
		0.20	0.23	0.21	0.22	0.27	0.27	0.20	0.18	0.21	0.19	0.13	0.21	0.19	0.14	0.10	0.21	0.20	0.21
		0.48	0.41	0.45	0.44	0.33	0.34	0.48	0.53	0.45	0.51	0.65	0.44	0.50	0.62	0.73	0.44	0.48	0.44

Fold change total	15	15	15	15	15	15	15	15	15	15	15	15	15	15	15	15	15	15

OHP = 1-hydroxypyrene, ΣOH-Fluo = sum of fluorene metabolites, ΣOH-Nap = sum of naphthalene metabolites, ΣOH-Phen = sum of phenanthrene metabolites

Table S3-8. Log $K_{\text{SWB-air}}$ values from previous studies and the present study. Both coefficients are ratios of PAH concentrations between SWBs and air. Estimated changes in the coefficients between silicone and air per degree increase in temperature were derived from the linear trendlines shown in Figure 3-. Blank cells are due to missing data from previous studies, and/or values being below the detection limit.

	Log $K_{\text{SWB-Air}}$				
PAH	**Anderson et al. (2017)**	**Donald et al. (2019)**	**This study, Jacket SWBs**	**This study, Wrist SWBs**	**Estimated change in log K per degree increase in temperature**
Naphthalene	5.9	5.3	3.3	2.1	-0.29
Acenaphthylene	6.2	6.8	3.4	1.6	-0.40
Acenaphthene	6.3	6.6	3.7	2.0	-0.35
Fluorene	6.4	7.2	3.6	2.0	-0.41
Phenanthrene	6.7	8	3.4	1.8	-0.49
Anthracene	6.6	7.9	3.4	1.6	-0.49
Fluoranthene	7.2	9.8	3.1	1.9	-0.68
Pyrene	7	9.3	3.0	1.7	-0.65
Benz[*a*]anthracene		10.4	2.5		
Chrysene		10.9	2.4		
Benzo[*b*]fluoranthene		12	2.4	2.1	
Benzo[*k*]fluoranthene		12.5	1.9		
Benzo[*a*]pyrene		12.6	2.3	2.2	
Indeno[1,2,3-*cd*]pyrene		13.5	2.2		
Dibenz[*a,h*]anthracene		13.8	2.4	2.1	

Chapter 4 Effectiveness of reducing firefighters' exposure to PAHs and genotoxins by implementing dermal cleaning interventions

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Study Design and Application for Funding	Jennifer LA Keir Jules M Blais Paul A White Tracy L. Kirkham Rocio Aranda-Rodriguez
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Sample Collection	Jennifer LA Keir
Chemical Analyses	Jennifer LA Keir
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Abstract

Firefighters are exposed to carcinogenic and mutagenic combustion emissions, including polycyclic aromatic hydrocarbons (PAHs). Fire service and firefighter cancer advocacy groups recommend skin cleaning using wipes or washing with detergent and water after exposure to smoke, although these strategies have not been proven to reduce exposures to harmful combustion products like PAHs. This study assessed dermal decontamination methods to reduce PAH exposures by firefighters participating in live fire training scenarios. Study participants (n=88) were randomly assigned to an intervention group (i.e., two types of commercial skin wipes, detergent and water, or a control group who did not use any skin decontamination). PAHs were measured in personal air (during the fire) and dermal wipe samples (before and after fire suppression, and after dermal decontamination). PAH metabolites and mutagenicity were measured in urine samples before and after fire suppression. Airborne PAH concentrations during the fire ranged between 200 and 3969 µg/m3 (mean= 759 µg/m^3, SD=685 µg/m^3). Firefighters had higher total PAHs and high molecular weight PAHs on their skin after the fire compared to before (1.3- and 2.2-fold, respectively, p<0.01). Urinary PAH metabolites increased significantly following exposure to the training fires by 1.7 – 2.2-fold (depending on the metabolite, p<0.001). Urinary mutagenicity did not differ significantly. Detergent and water was the only intervention that removed a significant amount of total PAHs from the skin (0.72 ng/cm^2 pre-intervention vs. 0.38 ng/cm^2 post-intervention, p<0.01). However, fold changes in urinary PAH metabolites (i.e., pre- versus post-exposure levels) did not differ between any of the dermal decontamination methods or the control group. These data suggest that despite on-site attempts to remove PAHs from firefighters' skin, the examined interventions did not reduce

the internal dose of PAHs. Future work should investigate preventing initial exposure using other interventions, such as improved personal protective equipment.

Introduction

Firefighters face serious occupational hazards, including heat, stress (physical/mental), and toxic chemicals. Epidemiological studies have shown that these exposures can have lasting effects on firefighters, resulting in an increased risk of cancer and other serious diseases compared to the general population (Jalilian et al. 2019, Soteriades et al. 2019, Casjens et al. 2020). In fact, the International Agency for Research on Cancer has classified the occupation of firefighting as ‘carcinogenic’ (i.e., Group 1) (Demers et al. 2022). Exposures to carcinogens can occur from firefighters’ inherent contact with combustion emissions that can contain benzene, chloroform, acrolein, formaldehyde, asbestos, lead, and polycyclic aromatic hydrocarbons (PAHs) (Bolstad-Johnson et al. 2000, Golka and Weistenhöfer 2008). PAHs are also mutagenic and teratogenic and are of particular concern due to their ubiquitous formation during combustion events (IARC 2010).

Several studies have assessed firefighters’ exposures to PAHs by measuring PAHs in air, on skin, and on PPE/clothing (Keir et al. 2020, Banks et al. 2021a, 2021b). For example, Keir et al. (2020) found air PAH concentrations sometimes exceeded occupational exposure limits and that firefighters’ experienced significant increases of PAHs on skin and PPE. Urinary PAH metabolites have also been broadly used to assess PAH exposure and uptake (i.e., internal dose) and several studies have found increased PAH metabolites associated with suppression of emergency municipal fires (Keir et al. 2017, Hoppe-Jones et al. 2021), training fires (Wingfors et al. 2018), staged fires (Fent et al. 2020), and wildfires (Cherry et al. 2021). Urinary mutagenicity is another non-invasive tool that permits a non-targeted exposure assessment to mutagenic aromatic compounds excreted via urine; it does not require *a priori* information on the identity of the putative toxicants. Increases in urinary mutagenicity have been measured in firefighters engaged in emergency fire suppression (Keir et al. 2017).

Several researchers have noted that dermal penetration is likely the predominant PAH exposure pathway (Laitinen et al. 2012, Fent et al. 2014, Keir et al. 2017). Firefighter's airways are protected by their self-contained breathing apparatus (SCBA), whereas skin is typically exposed to combustion emissions, and elevated temperatures encountered during fire suppression can enhance skin permeability (Chang and Riviere 1991, Jones et al. 2003, Park et al. 2008). In fact, several studies have found significantly elevated post-firefighting levels of PAHs on firefighters' skin, such as on the neck (Fent et al. 2014, Keir et al. 2017). Given the weight of evidence above that firefighters are exposed to PAHs through their skin, post-exposure removal of dermally-deposited PAHs could potentially reduce overall exposure and internal dose (Fent et al. 2014, 2017, Fernando et al. 2016, Keir et al. 2017). As such, dermal decontamination has been suggested as an effective means of removing PAHs deposited on skin. Indeed, skin cleaning wipes are now often deployed for post-fire suppression dermal decontamination. The intention is to reduce post-exposure dermal absorption of carcinogens via on-scene removal of contaminants deposited on skin, i.e., prior to showering upon return to the fire station. However, the efficacy of dermal cleaning procedures for reduction of PAH exposures and internal dose has not been broadly investigated. Fent et al. (2017) found that baby wipes were able to significantly reduce post-exposure PAH levels on firefighters' necks by a median of 54%. However, they only considered external PAH contamination levels; they did not consider PAH absorption and internal dose (e.g., urinary metabolite concentration). Moreover, skin-cleaning wipes can contain a range of ingredients, and it is not clear whether the specific types of wipes and/or wiping procedures can alter the ability to remove PAHs deposited on skin. This study aimed to determine whether skin decontamination procedures (i.e., select skin wipes, detergent and water) reduce PAHs deposited on skin and internal dose (i.e., urinary PAH metabolites and mutagenicity). The study results will permit firefighters and decision-makers to make evidence-based choices regarding the most appropriate way to reduce dermal exposures to combustion emissions.

Materials and Methods

Study design

Research ethics approval was obtained from the University of Ottawa Research Ethics Board (i.e., H-08-18-867) and Health Canada's Research Ethics Board (i.e., REB 2018-0020).

Firefighters from the Ottawa Fire Services and the Canadian Armed Forces were recruited via email or during classroom training sessions. Eligible participants were non-smokers who did not live with smokers, who agreed to avoid consumption of charbroiled foods and non-occupational exposures to combustion sources for the duration of enrollment, and did not participate in fire suppression activities in the previous 72 hours. Each participant completed a detailed questionnaire about their personal habits, overall health, and the nature of their employment (i.e., duration, secondary employment, etc.).

Sampling was conducted during particle board training fires in modified shipping containers (40' length x 8' width x 8.5' height) since they are controlled environments where multiple people are exposed under similar conditions. The shipping containers could hold 6-8 firefighters at a time. The training involved instructors near the fire at one end of the shipping container, controlling the fire's growth. The other firefighters, including the study participants, were grouped in the same container in a static position, moving occasionally in a circular pattern to take turns applying water on the fire with a nozzle. Activity, fuel load, and conditions were identical for all fires to ensure equivalent exposure.

Firefighters were randomly assigned to one of four post-fire decontamination methods on the day of sampling with a minimum of four participants recruited per fire (i.e., one per decontamination group). A second set of participants could be sampled (i.e., maximum of eight firefighters) per fire.

The decontamination methods included:

1. **Commercial wipe A**: premoistened, non-rinse, 7 x 7 inch disposable cloths marketed for cleaning of skin after exposure to substances such as grease or paint. The first two listed ingredients were water and an alcohol.
2. **Commercial wipe B**: premoistened, fragrance free baby wipes. The first two listed ingredients were water and aloe vera extract.
    - The two commercial wipes ranged in cost and ingredients.
3. **Detergent and water:** applied using a washcloth dipped in a bucket with an approximately 4 tablespoons of dish detergent in 5 L of water. This mixture was chosen due to its effectiveness at removing PAHs from contaminated gear (Fent et al. 2017).
4. **Control group**: no dermal decontamination was conducted.

Those assigned to a decontamination method were instructed to wash their skin in the same way they would after removing their PPE, including cleaning their arms, legs, neck, face, and hands. Decontamination occurred immediately upon exiting the fire and removal of their PPE (i.e., <10 minutes). Each decontamination group (i.e., three types of dermal cleaning and the control) contained 22 individuals.

Skin wipe samples

Skin PAH concentrations were assessed using a previously employed methodology (Keir et al. 2017). Briefly, skin wipe samples were collected using AlphaWipes® (Texwipe Inc., Kernersville, NC, USA) pre-wetted with 70% isopropyl alcohol. A 5 x 6 cm template was used to collect skin wipe samples from the forehead, neck, and wrist. Wipe samples were taken on the left side of the forehead and neck, and left wrist for the pre- and post-fire samples. After decontamination, wipe samples were taken on the right side of the forehead and neck, and right wrist to ensure removal of contaminants was strictly from the decontamination and not from previous skin surface sampling. The three wipes (i.e., neck, forehead, and wrist) were pooled together as one sample for each sampling period: pre-fire, post-fire, and post-decontamination.

Air samples

Personal air samples of at least one participant per fire were collected using the methodology employed previously (Keir et al., 2017). Briefly, GilAir Plus pumps (Levitt Safety, Ottawa, ON) ran at 2.5 L/min with a polyurethane foam (PUF) cartridge (URG, Chapel Hill, NC) and a QM-A 25 mm quartz filter (Whatman, Maidstone, UK). The sampling pump was placed in the inside pocket of the PPE coat and connected to the sample collection cartridge using polypropylene tubing along the inside of the coat. The sample collection cartridge was affixed to the back of the collar of the Bunker gear coat with a hook-and-loop fastener. Samples were collected starting immediately before entering fire for the full duration of the fire and stopped upon exit of the structure. Pumps were calibrated before sampling using a Gilian Gilibrator-2 calibrator (Levitt Safety, Ottawa, ON) for quality control. Field and travel blanks were also collected and are further described in the supplementary information.

Air and wipe PAH analyses

The concentrations of the US EPA 16 priority PAHs, plus a few others, were determined for skin surface and air samples at the Laboratory for the Analysis of Natural and Synthetic Environmental Toxicants (LANSET), University of Ottawa. Air samples were analysed as previously described in Keir et al. (2020). Briefly, all samples were spiked with a recovery standard of 5 deuterated PAHs (i.e., naphthalene-d_8, acenaphthene-d_8, phenanthrene-d_{10}, benzo(*a*)anthracene-d_{12}, and perylene-d_{12}). (Cambridge Isotope Laboratories Inc., Tewksbury, MA). Air samples were extracted with dichloromethane using accelerated solvent extraction (ASE 200, Dionex Corporation, Sunnyvale, CA). Skin surface wipe samples were extracted via sonication twice with 15 mL of 3:1 hexane:acetone. Extracts from pooled wipes were evaporated to approximately 30 mL. HPLC grade water was added, and the organic layer decanted off. Method blanks for both sample types were extracted following the same procedures. Extracts were concentrated under a gentle nitrogen stream to approximately 1 mL in 2,2',4-trimethylpentane before being spiked with an internal standard (p-terphenyl-d_{14}, Cambridge Isotope Laboratories, Tewksbury, MA). Samples were analysed by gas-chromatography (Agilent 7890B)-mass spectrometry (Agilent 5977B, Agilent Technologies, Santa Clara, CA, USA) on an HP-5MS UI 60 m, 0.25 µm, 250 µm column with selective ion monitoring. PAH concentrations were method blank corrected, then recovery corrected using the deuterated PAHs added to the samples at the time of extraction. High and low molecular weight (MW) PAHs were calculated using the sum of PAHs with 4 or more rings or 3 or less rings, respectively.

Urine sampling & analyses

Spot urine samples were collected in sterile 120 mL polypropylene containers prior to fire training. Participants were instructed on proper technique to prevent contamination of the specimen. Post-fire urine samples were collected for 18-hours after fire training in 3 L Urisafe 24-hr urine collection containers (Simport Scientific, Saint-Mathieu-de-Boleil, QC) to collect most of the PAH metabolites excreted pertaining to the fire exposure (i.e., 3+ half-lives). Subjects were instructed to keep the 18-hr urine samples in the fridge. Spot urine samples were stored chilled until transported to the University of Ottawa where aliquots were stored at -20°C until analysis.

Urine aliquots were sent to the ISO/IEC 17025 and ISO/IEC 17043 accredited Human Toxicology Laboratory of the National Institute of Public Health of Quebec (INSPQ) (Quebec City, QC, Canada) for analysis of urinary PAH metabolites via GC-MS/MS (Gaudreau et al. 2016). Briefly, urinary metabolites were deconjugated with β-glucuronidase in pH 5.0 sodium acetate buffer, extracted twice with hexane, and derivatized with N-methyl-N-(trimethylsilyl) trifluoroacetamide (MSTFA). Samples were spiked with 25 µL of an internal standard solution (1-methoxyfluorene 50 µg/L in benzene) prior to injection. 19 urinary PAH metabolites were measured: 1-hydroxynaphthalene, 2-hydroxynaphthalene, 2-hydroxyfluorene, 3-hydroxyfluorene, 9-hydroxyfluorene, 1-hydroxyphenanthrene, 2-hydroxyphenanthrene, 3-hydroxyphenanthrene, 4-hydroxyphenanthrene, 9-

hydroxyphenanthrene, 3-hydroxyfluoranthene, 1-hydroxypyrene, 1-hydroxybenz[*a*]anthracene, 3-hydroxybenz[*a*]anthrancene, 2-hydroxychrysene, 3-hydroxychrysene, 4-hydroxychrysene, 6-hydroxychrysene, and 3-hydroxybenzo[*a*]pyrene.

Urinary mutagenicity was measured using methodology employed previously (Keir et al. 2017). The Ames−Salmonella reverse mutation assay (i.e., the Ames test) was employed using *Salmonella typhimurium* strain YG1041 provided by Dr. Takehiko Nohmi (NIHS, Tokyo, Japan) in the presence of an exogenous metabolic activation mixture containing Aroclor-induced rat liver extract (Molecular Toxicology Inc., Boone, NC). Urine samples were filtered, enzymatically deconjugated and concentrated using C18 solid-phase extraction with methanol elution. Five doses were tested; second assays were run with doses in the linear response range for initially positive assays. A simultaneous positive control (i.e., 0.075 μg/plate 2-aminoanthracene, Molecular Toxicology Inc.) and negative solvent control (i.e., dimethyl sulphoxide) were examined to ensure assay performance on each test day. Samples were incubated at 37 °C for 72 h before the frequency of revertant (rev) colonies were scored. Mutagenic potency was calculated as the slope of the initial linear portion for samples that had a significant concentration−response ($p<0.05$). Samples without a significant concentration-response were given a value of zero. Mutagenic potency is expressed as rev/mmol of solute.

Urinary creatinine and osmolality concentrations were measured to correct for urinary dilution. Creatinine was measured using a Cayman Chemical (Ann Arbor, MI, USA) colorimetric assay kit (No. 500701), and osmolality using a VAPRO® Vapor Pressure Osmometer (Model 5600). Osmolality was employed to correct for urinary dilution and kidney function; osmolality-correction has been shown to provide more robust adjustment of hydration compared to creatinine (Middleton et al. 2016).

Data analyses

PAHs, or PAH metabolites, with greater than 95% of samples below the detection limit were omitted from analyses and PAH sum determinations. If <20% of the values for a PAH were below the detection limit, non-detects were replaced with the limit of detection divided by the square root of 2. If more than 20% of the samples for a particular PAH were below the detection limit, non-detects were replaced with values calculated using Robust regression on order statistics using NDExpo Version 1.0 (http://expostats.ca/site/app-local/NDExpo/). The data were log-transformed where appropriate based on visual inspection of box and Q-Q plots, and results of a Shapiro-Wilk test for normality. A paired t-test was employed to investigate the effect of fire suppression (i.e., pre- and post-fire) for urinary PAH metabolites, urinary mutagenicity, and skin wipes, and the effect of dermal decontamination (i.e., post-fire and post-decontamination differences) for skin wipes. Analysis of variance (ANOVA) with post-hoc multiple range testing (i.e., Duncan method) was used to determine mean exposure differences between the decontamination methods. Data were analyzed using SAS OnDemand for Academics (Release 3.8, SAS Institute, Cary, NC, USA). Differences were considered statistically significant when $p < 0.05$.

Results

Samples were collected from 88 firefighters during 21 training fires held on 8 days between October 2018 and November 2019. Participants (6 females, 82 males) were, on average, 34.4 years old and had 5.5 years of service. Details of participants' demographics can be found in Table 4-1.

Table 4-1 Self-reported participant demographic information (N=88).

		N	Mean (5th-95th percentile)
Gender	Male	82	
	Female	6	
Age			34.4 (33.5-49.0)
Years as a firefighter			5.5 (5-18.8)
Current title	Firefighter	82	
	Fire inspector	4	
	Lieutenant	1	
	Captain	1	
Age at start of firefighting career			26.6 (26-38)
Second job	Yes	35	
	No	53	
Fitness level	Excellent	8	
	Very good	40	
	Good	35	
	Fair	4	
	Poor	1	
Average times a week of 30 minutes or more of continuous exercise activity outside of work	<1	1	
	1-2	14	
	2-3	14	
	3-4	38	
	>5	17	
Overall health	Excellent	14	
	Very good	43	
	Good	30	
	Fair	1	
	Poor	0	
Weight	Overweight	13	
	Just about right	74	
	Underweight	1	

Personal air samples

Twenty-seven personal air samples were collected (i.e., some fires included more than one participant wearing an air sampler). One sample was omitted from analyses due to technical difficulties. Air samples averaged a duration of 43.9 minutes (ranging from 29.7 to 61.7 minutes). The mean of total PAHs in air samples was 759 µg/m^3 and ranged from 200 to 3970 µg/m^3. Naphthalene made up the largest proportion of the total PAHs followed by acenaphthylene and phenanthrene (Table 4-2). The average proportion of the known human carcinogen benzo[*a*]pyrene in air samples was 0.7% (SD=0.4%, range=0.2-1.5%). Probable/possible carcinogenic PAHs (i.e., naphthalene, benz[*a*]anthracene, chrysene, benzo[*b*]fluoranthene, benzo[*k*]fluoranthene, indeno[1,2,3-*cd*]pyrene, and dibenz[*a*,*h*]anthracene) made up on average 51.2% (SD=22.1%, range=13.6-79.2%) of measured PAHs. Results for air sample field and travel blanks can be found in Tables S4-1 and S4-2.

Table 4-2 Concentration of PAHs in personal air samples (N=27) collected during fire training activities in µg/m^3. Each fire training scenario included at least one individual wearing a personal air sampler; not every participant wore a personal air sampler.

PAH	Mean	Std Dev	Min	Max	Detection frequency
Naphthalene	390.26	463.95	21.17	2502.27	100%
Acenaphthylene	141.94	117.93	31.70	568.46	100%
Acenaphthene	9.61	6.97	1.91	35.68	100%
Fluorene	26.43	18.96	5.81	97.16	100%
Dibenzothiophene	0.49	1.28	<LOD	6.58	96%
Phenanthrene	77.26	66.19	21.52	350.67	100%
Anthracene	20.00	13.98	4.21	73.41	100%
Fluoranthene	23.76	16.85	6.39	91.02	100%
Pyrene	29.83	23.51	7.10	108.17	100%
Retene	5.52	2.76	1.75	12.85	100%
Benz[*a*]anthracene	6.11	4.98	1.42	25.96	100%
Chrysene	6.85	5.39	1.62	28.39	100%
Benzo[*b*]fluoranthene	3.44	2.80	0.69	14.54	100%
Benzo[*k*]fluoranthene	4.49	3.72	1.05	19.77	100%
Benzo[*e*]pyrene	2.88	2.37	0.69	12.39	100%
Benzo[*a*]pyrene	5.29	4.50	1.17	23.30	100%
Indeno[1,2,3- *cd*]pyrene	2.76	2.44	<LOD	12.26	96%
Dibenz[*a,h*]anthracene	0.30	0.29	<LOD	1.48	93%
Benzo[*ghi*]perylene	2.21	2.00	<LOD	10.19	96%
Total PAHs	759.44	685.39	200.87	3969.40	

<LOD= below limit of detection

Skin surface samples

Concentrations of PAHs from skin surface samples collected pre-fire, post-fire, and post-decontamination methods, stratified by decontamination method, are reported in Table S4-3. Total PAHs and high molecular weight PAH skin exposures significantly increased after fires (1.3- and 2.2-fold increase, $p<0.01$ and < 0.0001, respectively). Participants who decontaminated with detergent and water had significantly higher skin exposures after fire exposure ($p<0.05$) compared to the other groups (Figure S4-1). However, no significant differences were observed when naphthalene was removed from the total PAH concentrations. Fold change in PAH skin exposures following decontamination (i.e., concentration after decontamination/skin concentration before decontamination after fire exposure) shows that detergent and water removed the most PAHs, where decreases in all 14 individual PAHs with reportable levels were observed, 10 of them decreasing significantly and six significantly lower than the control group (Table 4-3). To determine which decontamination method was most effective, fold-change decreases in PAHs after decontamination (i.e., compared to post-fire) between the intervention groups were also compared. Intervention groups that had fold-change decreases significantly different than the control group are highlighted in Table 4-. Overall, compared to the control group, detergent and water removed significantly more total PAHs, high MW PAHs, acenaphthylene, fluoranthene, pyrene, retene, benz[*a*]anthracene, and chrysene. Wipe B was also significantly better than the control at removing high molecular weight PAHs and retene. Interestingly, the control had a higher fold-change decreases in naphthalene compared to participants using Wipe A; in fact, participants using Wipe A had, on average, higher levels of naphthalene on their skin post-decontamination compared to before.

Table 4-3 Fold-change decreases post-decontamination compared to post-fire in PAHs on skin surface samples (N=22). Fold-change was calculated using the skin concentration after decontamination divided by the skin concentration before decontamination following fire exposure. Values below 1 signify a decrease in skin exposure levels after decontamination compared to before. Values are bolded when significantly different from control ($p<0.05$). * and ** signify when post-decontamination is significantly lower than post-fire as determined by a paired t-test, $p<0.05$ and $p<0.01$, respectively.

	Wipe A				**Wipe B**				**Detergent & Water**				**Control (no decon.)**			
	GM	**SE**	**5Pctl**	**95Pctl**	**GM**	**SE**	**Min**	**Max**	**GM**	**SE**	**Min**	**Max**	**GM**	**SE**	**Min**	**Max**
Individual PAHs																
Naphthalene	**1.59**	0.47	0.16	18.10	0.91	0.14	0.23	2.69	0.61*	0.13	0.10	1.30	0.54	0.21	0.01	4.90
Acenaphthylene	1.26	0.40	0.15	14.17	0.92	0.22	0.07	4.09	**0.45****	0.11	0.04	1.83	1.42	0.52	0.04	25.35
Acenaphthene	2.29	1.17	0.12	202.50	0.92	0.31	0.03	3.48	0.80**	0.28	0.03	2.74	1.01	0.33	0.04	5.65
Fluorene	0.67	0.34	0.01	5.57	0.66	0.33	0.01	1.35	0.35	0.22	0.00	11.01	0.94	0.44	0.00	7.85
Dibenzothiophene	1.05	0.69	0.00	13.29	0.89	0.56	0.00	3.58	0.74	0.44	0.00	3.06	1.60	0.90	0.02	112.98
Phenanthrene	0.75	0.30	0.02	7.81	0.56	0.24	0.01	1.30	0.36*	0.17	0.00	17.01	0.94	0.40	0.01	21.01
Anthracene	0.59	0.26	0.01	5.66	0.89	0.42	0.01	21.78	0.38*	0.14	0.01	1.50	1.22	0.59	0.01	21.57
Fluoranthene	1.17	0.32	0.28	11.84	0.55	0.20	0.01	2.63	**0.35***	0.17	0.00	12.56	1.12	0.36	0.13	6.02
Pyrene	0.76	0.25	0.03	6.73	0.49	0.19	0.01	6.80	**0.29****	0.10	0.01	1.33	1.10	0.31	0.13	5.78
Retene	0.97	0.19	0.27	3.26	**0.56**	0.16	0.01	2.16	**0.41****	0.11	0.02	1.55	1.37	0.39	0.09	23.80
Benz[*a*]anthracene	0.92	0.35	0.04	30.95	0.44	0.26	0.01	32.49	**0.28****	0.10	0.02	3.26	1.57	0.75	0.02	52.25
Chrysene	0.76	0.20	0.09	3.77	0.70	0.34	0.01	11.04	**0.27****	0.09	0.01	1.44	0.99	0.42	0.02	24.15
Benzo[*b*]fluoranthene	1.00	0.23	0.10	7.13	0.99	0.25	0.08	7.71	0.97	0.15	0.13	1.43	1.40	0.44	0.10	12.32
Benzo[*ghi*]perylene	1.55	0.54	0.10	28.85	1.57	0.37	0.12	7.28	0.88	0.24	0.14	7.42	1.86	0.61	0.12	18.86
PAH groupings																
LMW PAHs	1.03	0.22	0.16	5.60	0.81	0.12	0.17	2.03	0.58**	0.10	0.08	1.37	0.81	0.22	0.03	5.14
HMW PAHs	0.84	0.12	0.31	2.37	**0.61***	0.13	0.09	1.47	**0.45****	0.09	0.05	1.52	1.11	0.21	0.20	5.45
Total PAHs	1.02	0.19	0.25	5.49	0.77	0.12	0.16	1.68	**0.53****	0.07	0.10	1.34	0.93	0.21	0.08	3.89

GM: geometric mean; SE: standard error; LMW = low molecular weight; HMW = high molecular weight.

Benzo[*a*]pyrene, benzo[*e*]pyrene, benzo[*k*]fluoranthene, indeno[1,2,3- *cd*]pyrene, and dibenz[*a*,*h*]anthracene were >97% below detection thus were omitted from analyses.

Additional analyses were conducted to investigate the removal of the carcinogenic PAHs. Three possibly carcinogenic PAHs were measured in detectable amounts (i.e., chrysene, benz(*a*)anthracene, and benzo(*b*)fluoranthene). The fold change in the sum of the possibly carcinogenic PAHs for post-fire compared to post-decontamination skin wipes were calculated; the results obtained revealed that the three dermal decontamination intervention groups had significantly different fold changes compared to the control (i.e., removed more possible carcinogens compared to the control) ($p<0.05$). Additional analyses investigated differences in skin wipe concentrations of the possibly carcinogenic PAHs post-fire compared to post-decontamination. Detergent and water removed a significant amount of possibly carcinogenic PAHs (i.e., mean difference of 0.01 ng/cm^2, $p<0.001$); Wipe A results indicated a weak, significant difference (i.e., mean fold change of 0.007 ng/cm^2, $p=0.045$).

Urinary PAH metabolites

Eight metabolites had interfering values or were below the detection limit in over 95% of the samples and were omitted from analyses (i.e., 1- and 3-hydroxybenz[*a*]anthracene, 2-, 3-, 4-, and 6-hydroxychrysene, 3-hydroxybenzo[*a*]pyrene, 3-hydroxyfluoranthene). The remaining 11 metabolites were grouped by their parent PAH (e.g., 1- and 2- hydroxynaphthalene reported as Σhydroxynaphthalenes). Background PAH metabolite levels (i.e., pre-fire samples) were not significantly different between participants who used different decontamination methods ($p>0.05$) (data not shown).

Significant increases in osmolality-corrected urinary PAH metabolites post-fire compared with pre-fire were found, ranging from 1.7 – 2.2-fold ($p<0.001$) (Figure 4-1). Significant increases were also observed in all four PAH metabolite groups for creatinine-corrected values (Table S4-8). No significant differences in urinary PAH metabolites were found between the three decontamination methods (p>0.05); both for osmolality-corrected (Table S4-7) and creatinine-corrected data (Table S4-8), or between the decontamination methods compared to the control (p>0.05, data not shown).

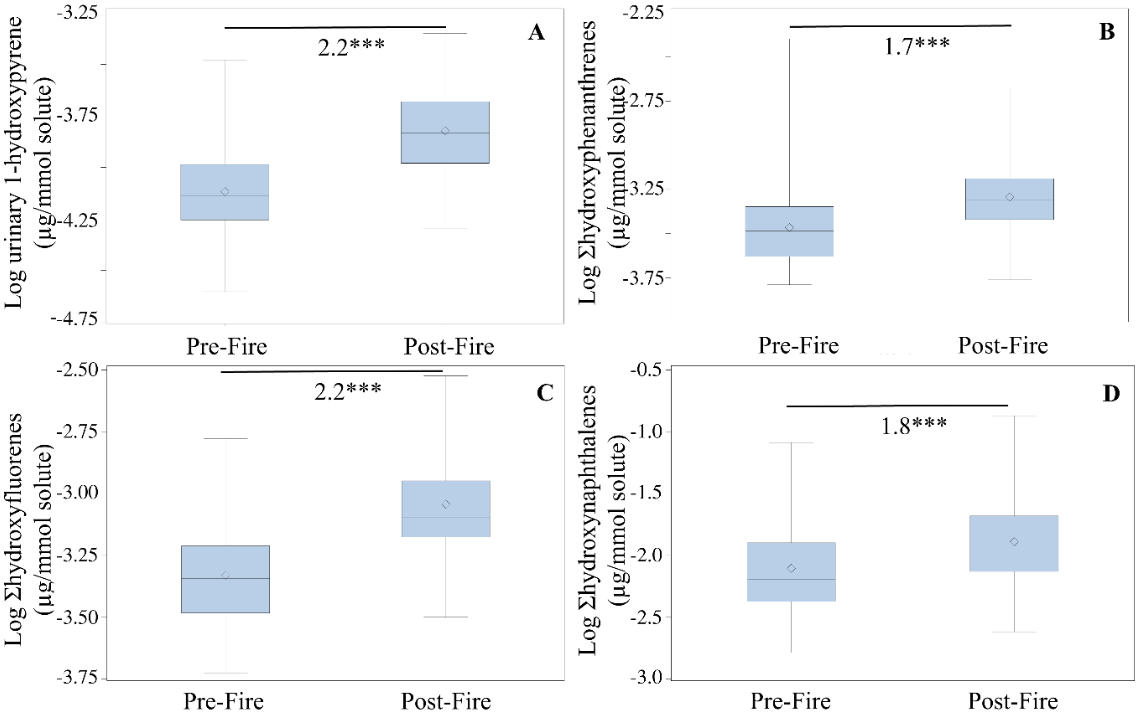

Figure 4-1. Boxplots illustrating osmolality-corrected urinary concentrations of (A) 1-hydroxypyrene, (B) Σhydroxyphenanthrenes, (C) Σhydroxyfluorenes, and (D) Σhydroxynaphthalenes in firefighters before live fire training (Pre-Fire) and after live fire training (Post-Fire). The box limits represent the interquartile range (i.e., 25th to 75th percentile), the diamonds represent the mean value, the solid line represents the group median, and the whiskers extend to the 5th and 95th percentiles. The asterisks (***) indicate a significant difference between pre- and post-fire levels at $p < 0.0001$ using a paired t-test.

Mutagenicity analyses

Three pre-fire urine samples had insufficient sample volume and one was too cytotoxic for bacteria to grow and were omitted from analyses. Only 29 of the 182 urine samples elicited a significant dose-response. No significant difference in urinary mutagenicity was found overall after fire exposure (n=172), for samples with a significant dose-response (n=29), nor for paired of pre- and post-fire samples with a significant dose-response (n=6). There were no significant differences in fold changes in urinary mutagenicity (i.e., pre- versus post-fire) between decontamination methods (data not shown).

Table 4-9 Calculation of absorption time for parent PAHs of the urinary metabolites measured.

PAH	Dermal absorption flux[a] (ng cm^{-2} $hour^{-1}$)	Post-fire skin concentration (ng/cm^2) (N=88) GM	Max	Calculated absorption time (seconds) for GM	for Max
Pyrene	3.01	0.026	0.12	31.10	148.21
Phenanthrene	4.02	0.05	1.09	44.78	979.18
Fluorene	22.16	0.009	0.35	1.46	57.53
Naphthalene	96.8	0.13	1.32	4.83	49.01

[a]From Silva et al. (2021)

Discussion

PAH air concentrations during fires provided a snapshot of the environment firefighters were exposed to during the training exercises. The results herein were similar to previous studies of air PAH concentrations during live fire training or simulated fires such as Kirk and Logan (2015) that reported concentrations between 430 μg/m^3 to 2,700 μg/m^3 and Fent et al. (2014) that reported concentrations under 2,000 μg/m^3 for one of the two sets of simulated fires they investigated. The other set of fires that Fent et al. (2014) investigated were markedly higher, ranging from around 2,000 to 12,000 μg/m^3. Banks et al. (2021) also reported higher air PAH concentrations than reported here, from live fire training using particleboard (i.e., 75,000 to 180,000 μg/m^3). Differences in air PAH concentrations between the various studies is likely a result of the fuel of the fires, fire temperatures and ventilation conditions (dependent on firefighting tactics), and role of the participant in fire suppression. Indeed, Banks et al. (2021) found firefighters' personal PAH concentrations to differ depending on the fuel of the training fire they attended (i.e., particleboard or diesel in a pan). Interestingly, air samples collected herein were notably higher than a previous study that collected personal air samples at emergency fire events, i.e., mean of 253 μg/m3 (Keir et al. 2020). The difference in means may be attributed to sample collection method; in the study of emergency fires, air samples were likely diluted as sample collection encompassed travel to and from the fire event. In contrast, sample collection for this study started immediately before entering the training fire and stopped immediately upon exiting the fire-containing structure. Additionally, participants in the present study were exposed while conducting interior fire suppression operations, whereas the study of emergency fires included firefighters at emergency fire events in multiple roles, both inside and outside structures. Keir et al. (2020) also reported higher proportions of benzo[a]pyrene (1% vs. 0.7%) and possible and probable carcinogenic PAHs (77% vs. 51%). The difference in PAH makeup may be a result of fuel differences: training fires utilize a single fuel type whereas emergency fires are composed of multiple fuel types and complex mixtures. This is consistent with a study by Banks et al. (2021) that presented data indicating higher proportions of possible and probable carcinogenic PAHs in personal air from fires with different fuels (i.e., particleboard fires versus diesel pan fires), calculated using their reported data.

The results obtained revealed that detergent and water was the most effective at removing PAHs from skin compared to two types of wipes or the control (i.e., no dermal cleaning). There are several reasons that may explain why detergent and water was the most effective. First, the surfactants and/or other ingredients in detergent and water may provide superior ability to remove PAHs from skin compared to the ingredients in the wipes (e.g., superior surfactants to remove the contaminants and/or the particles the contaminants are adsorbed to). Second, the textured material of the washcloths used in the detergent and water decontamination method may have provided superior removal of PAHs compared to the smooth, flat wipes. Third, although participants in the current study were allowed to use unlimited wipes or were given unlimited access to the bucket of detergent and water, unconscious bias may have occurred between wipes versus those using detergent and water, leading to less washing in those using wipes. Perhaps the familiarity of a washcloth led to spending more time on dermal decontamination compared to those using wipes. Indeed, research staff anecdotally noted that those using a washcloth tended to take longer to perform dermal decontamination compared to those that were given one of the two wipes. More research is required to determine if there is an optimal wipe formulation for removal of a significant amount of firefighters' post-fire dermal contamination. This could be useful for situations where detergent and water dermal decontamination is logistically not possible (e.g., during winter or in remote areas during wildland firefighting).

To the best of our knowledge, this is the first study to comparatively evaluate the ability of several post-fire dermal decontamination methods to remove PAHs from skin; moreover, to alter internal dose. Fent et al. (2017) is the only other study to have assessed the ability of post-fire dermal decontamination to remove PAHs from skin (but not internal dose). They found that baby wipes reduced PAHs on the neck of firefighters after firefighting by a median of 54%. Although the findings in Fent et al. (2017) are similar for the detergent and water intervention group in the present study, the two commercial wipes used for the intervention groups in this study reported lower removal rates of PAHs compared to the wipe used in Fent et al. (2017). The differences between the two studies are likely due to differences in the wipes used and the aforementioned reasons behind why various wipes or detergent and water protocols can differ in PAH removal efficiency. It is possible that other methods not yet investigated may be effective at removing dermally deposited PAHs and/or other organic mutagens, and should be investigated moving forward.

The lack of difference between urinary PAH metabolite increases between the intervention groups and the control group show that the post-fire dermal cleaning steps did not have a measurable effect on the internal dose of PAHs that firefighters received during the live fire training exercise. It is likely that post-fire dermal decontamination efforts are too late to prevent absorption into the body. This assertion is supported by the dermal absorption fluxes of the parent PAHs of the metabolites measured (i.e., pyrene, phenanthrene, fluorene, and naphthalene). Using post-fire skin PAH concentrations as a proxy for skin PAH concentrations during fire suppression, these fluxes suggest that all bioavailable pyrene, phenanthrene, fluorene, and naphthalene dermally deposited on firefighters in this study would be absorbed within seconds to minutes (Table 4). Furthermore, since firefighters experience increased skin temperatures while fighting fires (Horn et al. 2018), which has been shown to increase skin permeability (Park et al. 2008), the absorption of PAHs during firefighting is likely faster than reported flux rates. This suggests that absorption of the parent PAHs would have occurred before the intervention was conducted. During the time prior to absorption and/or for contaminants that take longer to absorb, dermal cleaning may also increase absorption by hydrating the skin (Maibach 2021). This "wash-in" effect, where chemicals are absorbed more readily into the skin with washing, has been reported for different chemicals including benzo[a]pyrene, N,N-diethyl-m-toluamide (DEET), and 1,1,1-trichloro-2,2-bis(4-chlorophenyl)ethane (DDT) (Moody et al. 1994, 1995b, 1995a). In fact, Moody and Maibach (2006) reported that the wash-in effect may increase both cutaneous and general systemic toxicity; they recommend caution when practicing skin washing for substance removal. More specifically, the authors recommend understanding the mechanisms of absorption to identify interventions that decrease occupational exposure and relatedly, exposure-caused occupational disease, so as to avoid a wash-in effect. That being said, post-fire dermal cleaning may be effective at reducing high molecular weight (HMW) PAHs (and other slower absorbing compounds) as they have lower fluxes, taking longer to penetrate the skin and absorb into the body (Silva et al. 2021). It is possible that post-fire dermal cleaning reduced absorption and cross contamination of compounds left on the surface of the skin. However, since HMW PAHs are excreted via the feces rather than the urine, any differences in internal dose of HMW PAHs could not be measured (Motorykin et al. 2015). Future work should investigate the influence of dermal cleaning on internal dose of larger PAHs using analyses of blood or fecal samples.

No significant differences in urinary mutagenicity were seen, contrary to previous studies that found significant increases in urinary mutagenicity for firefighters attending emergency, residential fires and wildland prescribed burns (Keir et al. 2017, Wu et al. 2021). The lack of significant change in urinary mutagenicity observed here suggests that the firefighters were not exposed to levels of mutagens that could be detected using the Salmonella reverse mutation test (i.e., Ames test). The lower exposure could be due to different and/or lower amounts of fuel used at the fire (i.e., particleboard), temperature differences, shorter duration of fire suppression activity compared to previous studies, and/or their PPE and exposure prevention methods effectively working to reduce exposure to combustion-derived mutagens. It is also possible that exposure to larger mutagens (e.g., benzo(a)pyrene) were not detected due to their excretion through pathways other than urine (e.g., fecal excretion). Future work could scrutinise the lack of detection of mutagens in urine and assess situations where more substantial exposures may occur, such as staged burns with household contents or fires of longer duration, that provide higher exposures to mutagens that could be detected via the Ames test; moreover, any differences related to the use of various dermal decontamination methods could also be examined.

The results of the influence of dermal decontamination on skin surface contamination reported herein and by Fent et al. (2017) focused only on PAHs, yet firefighters can be exposed to hundreds of different chemicals and the efficacy of dermal decontamination can change with different compounds. It is important to utilize exposure biomonitoring proxies that encompass exposure to a range of compounds. Urinary mutagenicity remains a useful tool to determine overall exposure to mutagens without having a priori information on the identity of the combustion-derived mutagens in complex mixtures of combustion emissions and should continue to be utilized. Other exposure biomonitoring proxies should also be deployed that encompass exposure to a range of compounds. For example, Grashow et al. (2020) used untargeted analyses in serum samples from female firefighters and office workers in San Francisco and detected phthalate metabolites, phosphate flame-retardant metabolites, phenols, pesticides, nitro and nitroso compounds, and per- and polyfluoroalkyl substances. Other options for measuring the influence of dermal decontamination on exposure could include use of assays to examine, for example, induced mutations and/or chromosomal damage in blood cells and/or urine-derived epithelial cells. Careful consideration of toxicokinetics would be required to ensure proper timing of sample collection to see any possible influences of dermal decontamination on exposure and/or effect.

Conclusions

It has been suggested that removal of combustion-derived carcinogens from firefighters' skin might reduce exposure. Of the three on-site decontamination procedures examined in this study, dish detergent and water was much more effective than the two commercially available skin cleaning wipes and significantly removed approximately half of the PAHs deposited on skin. Despite the effectiveness of the various decontamination methods for removal of PAHs from the skin surface, these differences were not reflected in the measured PAH metabolites in urine. Therefore, the present data indicate that even early attempts to remove PAHs from firefighters' skin do not reduce the internal dose received, likely because PAHs traverse the skin prior to decontamination efforts. Rather than post-exposure efforts, future work should focus on preventing the initial exposure through improved compliancy in use and quality of PPE such as novel textiles and/or improved fit of PPE to prevent passage of combustion by-products onto skin.

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References

Banks APW, Thai P, Engelsman M, Wang X, Osorio AF, Mueller JF. 2021a. Characterising the exposure of Australian firefighters to polycyclic aromatic hydrocarbons generated in simulated compartment fires. Int J Hyg Environ Health. 231:113637.

Banks APW, Wang X, Engelsman M, He C, Osorio AF, Mueller JF. 2021b. Assessing decontamination and laundering processes for the removal of polycyclic aromatic hydrocarbons and flame retardants from firefighting uniforms. Environ Res. 194:110616.

Bolstad-Johnson DM, Burgess JL, Crutchfield CD, Storment S, Gerkin R, Wilson JR. 2000. Characterization of Firefighter Exposures During Fire Overhaul. Am Ind Hyg Assoc J. 61:636–641.

Casjens S, Brüning T, Taeger D. 2020. Cancer risks of firefighters: a systematic review and meta-analysis of secular trends and region-specific differences. Int Arch Occup Environ Health. 93:839–852.

Chang SK, Riviere JE. 1991. Percutaneous Absorption of Parathion in Vitro in Porcine Skin: Effects of Dose, Temperature, Humidity, and Perfusate Composition on Absorptive Flux. Toxicol Sci. 17:494–504.

Cherry N, Galarneau JM, Kinniburgh D, Quemerais B, Tiu S, Zhang X. 2021. Exposure and absorption of PAHs in wildland firefighters: A field study with pilot interventions. Ann Work Expo Heal. 65:148–161.

Demers PA, DeMarini DM, Fent KW, Glass DC, Hansen J, Adetona O, Andersen MH, Freeman LEB, Caban-Martinez AJ, Daniels RD, Driscoll TR, Goodrich JM, Graber JM, Kirkham TL, Kjaerheim K, Kriebel D, Long AS, Main LC, Oliveira M, Peters S, Teras LR, Watkins ER, Burgess JL, Stec AA, White PA, DeBono NL, Benbrahim-Tallaa L, Conti A de, Ghissassi F El, et al. 2022. Carcinogenicity of occupational exposure as a firefighter. Lancet Oncol. 23(8):985-986.

Fent KW, Alexander B, Roberts J, Robertson S, Toennis C, Sammons D, Bertke S, Kerber S, Smith D, Horn G. 2017. Contamination of firefighter personal protective equipment and skin and the effectiveness of decontamination procedures. J Occup Environ Hyg. 14:801–814.

Fent KW, Eisenberg J, Snawder J, Sammons D, Pleil JD, Stiegel MA, Mueller C, Horn GP, Dalton J. 2014. Systemic Exposure to PAHs and Benzene in Firefighters Suppressing Controlled Structure Fires. Ann Occup Hyg. 58:830–845.

Fent KW, Toennis C, Sammons D, Robertson S, Bertke S, Calafat AM, Pleil JD, Wallace MAG, Kerber S, Smith D, Horn GP. 2020. Firefighters' absorption of PAHs and VOCs during controlled residential fires by job assignment and fire attack tactic. J Expo Sci Environ Epidemiol. 30:338–349.

Fernando S, Gallea M, VandenEnden L, House R, Verma D, Shaw L, McCarry BE, Shaw D, Britz-McKibbin P. 2016. Evaluation of Firefighter Exposure to Wood Smoke During Training Exercises at Burn Houses. Environ Sci Technol. 50:1536–1543.

Gaudreau É, Bérubé R, Bienvenu J-F, Fleury N. 2016. Stability issues in the determination of 19 urinary (free and conjugated) monohydroxy polycyclic aromatic hydrocarbons. Anal Bioanal Chem. 408:4021–4033.

Golka K, Weistenhöfer W. 2008. Fire fighters, combustion products, and urothelial cancer. J Toxicol Environ Heal Part B. 11:32–44.

Grashow R, Bessonneau V, Gerona RR, Wang A, Trowbridge J, Lin T, Buren H, Rudel RA, Morello-Frosch R. 2020. Integrating Exposure Knowledge and Serum Suspect Screening as a New Approach to Biomonitoring: An Application in Firefighters and Office Workers. Environ Sci Technol. 54:4344–4355.

Hoppe-Jones C, Griffin SC, Gulotta JJ, Wallentine DD, Moore PK, Beitel SC, Flahr LM, Zhai J, Zhou JJ, Littau SR, Dearmon-Moore D, Jung AM, Garavito F, Snyder SA, Burgess JL. 2021. Evaluation of fireground exposures using urinary PAH metabolites. J Expo Sci Environ Epidemiol 2021 315. 31:913–922.

Horn GP, Kesler RM, Kerber S, Fent KW, Schroeder TJ, Scott WS, Fehling PC, Fernhall B, Smith DL. 2018. Thermal response to firefighting activities in residential structure fires: impact of job assignment and suppression tactic. Ergonomics. 61:404–419.

IARC. 2010. Some non-heterocyclic polycyclic aromatic hydrocarbons and some related exposures. IARC Monogr Eval Carcinog Risks Hum. 92:1–853.

Jalilian H, Ziaei M, Weiderpass E, Rueegg CS, Khosravi Y, Kjaerheim K. 2019. Cancer incidence and mortality among firefighters. Int J Cancer. 145:2639–2646.

Jones K, Cocker J, Dodd LJL, Fraser I. 2003. Factors affecting the extent of dermal absorption of solvent vapours: a human volunteer study. Ann Occup Hyg. 47:145–150.

Keir JLA, Akhtar U, Matschke DMJ, Kirkham TL, Chan HM, Ayotte P, White PA, Blais JM. 2017. Elevated Exposures to Polycyclic Aromatic Hydrocarbons and Other Organic Mutagens in Ottawa Firefighters Participating in Emergency, On-Shift Fire Suppression. Environ Sci Technol. 51:12745–12755.

Keir JLA, Akhtar US, Matschke DMJ, White PA, Kirkham TL, Chan HM, Blais JM. 2020. Polycyclic aromatic hydrocarbon (PAH) and metal contamination of air and surfaces exposed to combustion emissions during emergency fire suppression: Implications for firefighters' exposures. Sci Total Environ. 698:134211.

Kirk KM, Logan MB. 2015. Firefighting Instructors' Exposures to Polycyclic Aromatic Hydrocarbons During Live Fire Training Scenarios. J Occup Environ Hyg. 12:227–234.

Laitinen J, Mäkelä M, Mikkola J, Huttu I. 2012. Firefighters' multiple exposure assessments in practice. Toxicol Lett. 213:129–133.

Maibach HI. 2021. Dermal Decontamination and Percutaneous Absorption. CRC Press. 315–326.

Middleton DRS, Watts MJ, Lark RM, Milne CJ, Polya DA. 2016. Assessing urinary flow rate, creatinine, osmolality and other hydration adjustment methods for urinary biomonitoring using NHANES arsenic, iodine, lead and cadmium data. Environ Heal. 15:68.

Moody RP, Maibach HI. 2006. Skin decontamination: Importance of the wash-in effect. Food Chem Toxicol. 44:1783–1788.

Moody RP, Nadeau B, Chu I. 1994. In vitro dermal absorption of pesticides: VI. In vivo and in vitro comparison of the organochlorine insecticide DDT in rat, guinea pig, pig, human and tissue-cultured skin. Toxicol Vitr. 8:1225–1232.

Moody RP, Nadeau B, Chu I. 1995a. In vitrodermal absorption ofN,N-diethyl-m-toluamide (DEET) in rat, guinea pig, and human skin. Vitr Toxicol J Mol Cell Toxicol. 8:263–275.

Moody RP, Nadeau B, Chu I. 1995b. In vivo and in vitro dermal absorption of benzo[a]pyrene in rat, guinea pig, human and tissue-cultured skin. J Dermatol Sci. 9:48–58.

Motorykin O, Santiago-Delgado L, Rohlman D, Schrlau JE, Harper B, Harris S, Harding A, Kile ML, Simonich SLM. 2015. Metabolism and excretion rates of parent and hydroxy-PAHs in urine collected after consumption of traditionally smoked salmon for Native American volunteers. Sci Total Environ. 514:170–177.

Park JH, Lee JW, Kim YC, Prausnitz MR. 2008. The effect of heat on skin permeability. Int J Pharm. 359:94–103.

Silva J, Marques-da-Silva D, Lagoa R. 2021. Reassessment of the experimental skin permeability coefficients of polycyclic aromatic hydrocarbons and organophosphorus pesticides. Environ Toxicol Pharmacol. 86:103671.

Soteriades ES, Kim J, Christophi CA, Kales SN. 2019. Cancer Incidence and Mortality in Firefighters: A State-of-the-Art Review and Meta-Analysis. Asian Pac J Cancer Prev. 20:3221.

Wingfors H, Nyholm JR, Magnusson R, Wijkmark CH. 2018. Impact of Fire Suit Ensembles on Firefighter PAH Exposures as Assessed by Skin Deposition and Urinary Biomarkers. Ann Work Expo Heal. 62:221–231.

Wu CM, Warren SH, Demarini DM, Song CC, Adetona O. 2021. Urinary mutagenicity and oxidative status of wildland firefighters working at prescribed burns in a Midwestern US forest. Occup Environ Med. 78:315–322.

Supplementary Information

Air sample blank collection

To ensure robust air contamination values, field and travel blanks were also collected. Field blanks were collected to account for any extraneous contamination acquired during the sample collection period, i.e., unused air samplers maintained on-scene on the sampling days. Travel blanks were unused air samplers brought to/from the analytical laboratory alongside the used samplers. Travel blanks were not volume corrected since they did not involve use of the sampling pump; values are reported in ng per sampler. The results of the field and travel blanks are provided in Tables S1 and S2, respectively. Negligible amounts of PAHs (i.e., ~1%) were detected in field blank samples relative to personal air samples collected during training. Similarly, average concentrations of total PAHs in travel blank samples were 0.3% of post-training levels, i.e., average of 82763.8 ng per sampler (data not shown). Thus, the sample collection process did not appreciably contribute to the measured post-exposure PAH contamination levels.

Figure S4-1 Boxplots comparing the fold change in skin wipe results (i.e., post- versus pre-fire) by intervention for (A) total PAHs and (B) total PAHs excluding naphthalene. The box limits represent the interquartile range (i.e., 25th to 75th percentile), the diamonds represent the mean value, the solid line represents the group median, the whiskers extend to the 5th and 95th percentiles, and the circles are outliers. Boxes with the same letter are not significantly different from each other (p<0.05).

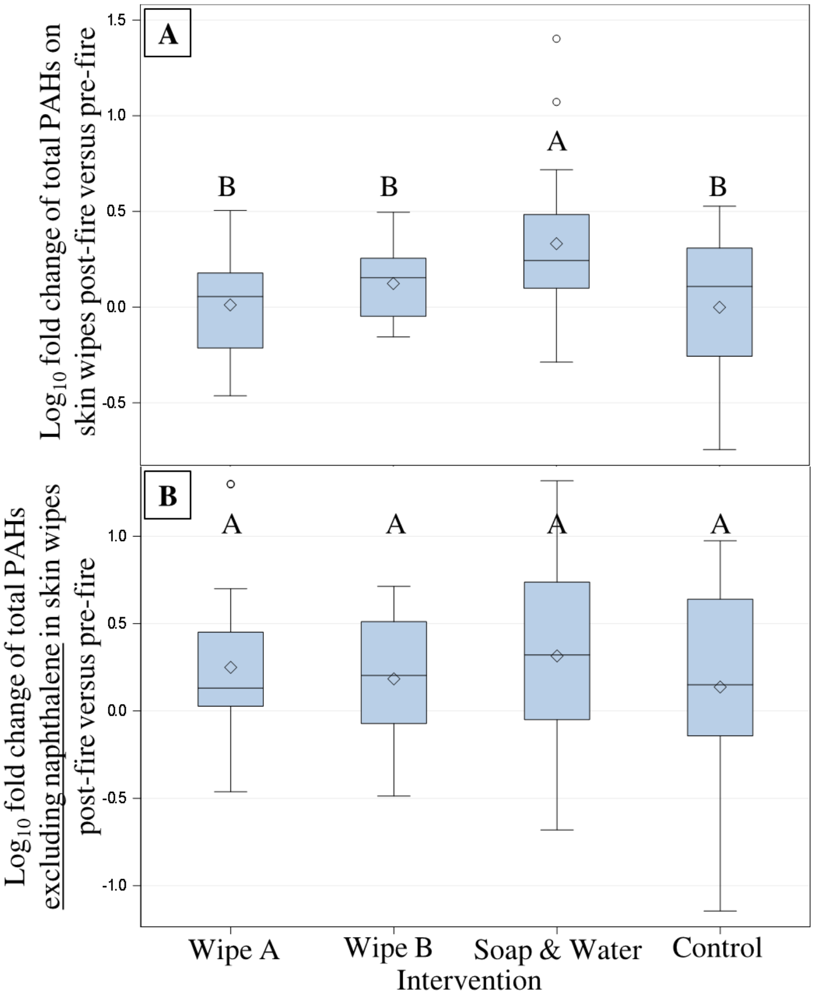

Table S4-1. PAH concentrations in air sample field blanks (N=3). Samples were collected on select sample collection days; values in µg/m^3.

PAH	Mean	Std Dev	Min	Max
Naphthalene	0.90	1.21	0.17	2.29
Acenaphthylene	0.89	1.12	0.21	2.19
Acenaphthene	BDL	-	BDL	0.57
Fluorene	0.71	0.85	0.21	1.70
Dibenzothiophene	0.18	0.18	0.07	0.39
Phenanthrene	1.91	2.00	0.66	4.22
Anthracene	0.15	0.15	0.05	0.31
Fluoranthene	0.47	0.58	0.12	1.14
Pyrene	2.04	2.59	0.52	5.02
Retene	0.10	0.13	0.02	0.25
Benz[*a*]anthracene	0.01	0.01	0.00	0.02
Chrysene	0.00	0.01	0.00	0.01
Benzo[*b*]fluoranthene	0.09	0.11	0.02	0.21
Benzo[*k*]fluoranthene	BDL	-	BDL	BDL
Benzo[*e*]pyrene	BDL	-	BDL	BDL
Benzo[*a*]pyrene	BDL	-	BDL	BDL
Indeno[1,2,3- *cd*]pyrene	BDL	-	BDL	BDL
Dibenz[*a*,*h*]anthracene	BDL	-	BDL	BDL
Benzo[*ghi*]perylene	BDL	-	BDL	BDL
Total PAHs	7.67	9.22	2.21	18.32

BDL=below detection limit

Table S4-2. PAH concentrations in air sampler travel blanks (N=7). Values in ng per sampler; sample collection pumps were off.

PAH	**Mean**	**Std Dev**	**Min**	**Max**
Naphthalene	24.09	12.73	BDL	34.77
Acenaphthylene	21.58	10.37	BDL	29.11
Acenaphthene	8.66	4.37	BDL	13.94
Fluorene	24.01	11.42	BDL	36.25
Dibenzothiophene	6.52	4.60	BDL	10.94
Phenanthrene	75.04	36.32	BDL	104.03
Anthracene	5.34	3.77	BDL	9.37
Fluoranthene	16.27	11.03	BDL	34.66
Pyrene	59.13	58.86	BDL	182.22
Retene	3.30	2.36	BDL	5.76
Benz[*a*]anthracene	0.67	0.76	BDL	1.84
Chrysene	2.20	3.04	BDL	7.85
Benzo[*b*]fluoranthene	1.64	1.38	BDL	3.28
Benzo[*k*]fluoranthene	BDL	BDL	BDL	2.48
Benzo[*e*]pyrene	BDL	BDL	BDL	0.75
Benzo[*a*]pyrene	BDL	BDL	BDL	1.33
Indeno[1,2,3-*cd*]pyrene	BDL	BDL	BDL	0.53
Dibenz[*a,h*]anthracene	BDL	BDL	BDL	BDL
Benzo[*ghi*]perylene	BDL	BDL	BDL	0.46
Total PAHs	249.53	123.93	76.93	438.84

BDL=below detection limit

Table S4-3 Summary of skin surface wipe PAH concentrations by intervention group. Values in ng/cm^2.

	Pre-fire				**Post-fire**				**Post-decontamination**			
PAH/PAH group	**GM**	**SE**	**Min**	**Max**	**GM**	**SE**	**Min**	**Max**	**GM**	**SE**	**Min**	**Max**
Wipe A												
Total PAHs	0.44	0.09	0.09	2.05	0.45	0.10	0.09	3.65	0.46	0.11	0.09	4.24
LMW PAHs	0.38	0.09	0.06	1.84	0.34	0.08	0.07	3.42	0.36	0.10	0.05	4.04
HMW PAHs	0.04	0.01	0.00	0.21	0.09	0.02	0.02	0.39	0.07	0.01	0.02	0.25
Naphthalene	0.23	0.05	<LOD	0.78	0.13	0.04	<LOD	1.32	0.21	0.06	<LOD	2.68
Acenaphthylene	0.01	0.00	<LOD	0.21	0.02	0.01	<LOD	0.45	0.03	0.01	<LOD	0.34
Acenaphthene	<LOD	0.00	<LOD	0.04	<LOD	<LOD	<LOD	0.10	0.01	0.00	<LOD	0.09
Fluorene	0.01	0.00	<LOD	0.23	0.01	0.00	<LOD	0.35	0.01	0.00	<LOD	0.27
Phenanthrene	0.03	0.01	<LOD	0.67	0.05	0.02	<LOD	1.09	0.04	0.01	<LOD	0.58
Anthracene	<LOD	0.00	<LOD	0.04	<LOD	<LOD	<LOD	0.07	<LOD	<LOD	<LOD	0.06
Fluoranthene	0.01	0.00	<LOD	0.10	0.02	0.01	<LOD	0.14	0.03	0.00	0.00	0.10
Pyrene	0.01	0.00	<LOD	0.11	0.03	0.01	<LOD	0.12	0.02	0.01	<LOD	0.11
Retene	<LOD	<LOD	<LOD	0.02	0.01	0.00	<LOD	0.09	0.01	0.00	<LOD	0.06
Benz[*a*]anthracene	<LOD	<LOD	<LOD	0.03	<LOD	<LOD	<LOD	0.05	<LOD	<LOD	<LOD	0.03
Chrysene	<LOD	<LOD	<LOD	0.03	<LOD	<LOD	<LOD	0.06	<LOD	<LOD	<LOD	0.03
Benzo[*b*]fluoranthene	<LOD	<LOD	<LOD	0.02	<LOD	<LOD	<LOD	0.03	<LOD	<LOD	<LOD	0.02
Benzo[*ghi*]perylene	<LOD	<LOD	<LOD	0.02	<LOD	<LOD	<LOD	0.02	<LOD	<LOD	<LOD	0.01
Wipe B												
Total PAHs	0.37	0.07	0.06	1.42	0.48	0.09	0.10	2.30	0.37	0.08	0.05	2.60
LMW PAHs	0.29	0.07	0.04	1.31	0.37	0.08	0.05	1.93	0.30	0.07	0.03	2.52
HMW PAHs	0.05	0.01	0.00	0.14	0.09	0.01	0.03	0.37	0.05	0.01	0.01	0.35
Naphthalene	0.17	0.05	<LOD	0.91	0.20	0.05	<LOD	0.97	0.19	0.05	<LOD	1.48
Acenaphthylene	0.01	0.00	<LOD	0.17	0.02	0.01	<LOD	0.20	0.02	0.01	<LOD	0.15
Acenaphthene	<LOD	<LOD	<LOD	0.04	0.01	0.00	<LOD	0.06	0.01	0.00	<LOD	0.05
Fluorene	0.01	0.00	<LOD	0.09	0.01	0.00	<LOD	0.20	<LOD	<LOD	<LOD	0.20
Phenanthrene	0.03	0.01	<LOD	0.26	0.03	0.01	<LOD	0.74	0.01	0.01	<LOD	0.59
Anthracene	<LOD	<LOD	<LOD	0.01	0.00	0.00	<LOD	0.06	<LOD	<LOD	<LOD	0.04
Fluoranthene	0.01	0.00	<LOD	0.05	0.02	0.01	0.00	0.13	0.01	0.00	<LOD	0.13
Pyrene	0.01	0.00	<LOD	0.05	0.02	0.01	<LOD	0.13	0.01	0.00	<LOD	0.13
Retene	<LOD	<LOD	<LOD	0.03	0.01	0.00	<LOD	0.04	<LOD	<LOD	<LOD	0.03
Benz[*a*]anthracene	<LOD	<LOD	<LOD	0.03	0.01	0.00	<LOD	0.03	<LOD	<LOD	<LOD	0.05
Chrysene	<LOD	<LOD	<LOD	0.03	<LOD	<LOD	<LOD	0.05	<LOD	<LOD	<LOD	0.04
Benzo[*b*]fluoranthene	<LOD	<LOD	<LOD	0.02	<LOD	<LOD	<LOD	0.02	<LOD	<LOD	<LOD	0.03
Benzo[*ghi*]perylene	<LOD	<LOD	<LOD	0.05	<LOD	<LOD	<LOD	0.02	<LOD	<LOD	<LOD	0.01
Detergent & Water												
Total PAHs	0.34	0.08	0.05	2.35	0.72	0.14	0.15	2.85	0.38	0.07	0.07	1.77

LMW PAHs	0.28	0.07	0.03	2.27	0.56	0.14	0.07	2.71	0.32	0.07	0.05	1.70
HMW PAHs	0.04	0.01	0.00	0.11	0.09	0.01	0.01	0.24	0.04	0.01	0.00	0.14
Naphthalene	0.18	0.04	<LOD	1.12	0.34	0.09	<LOD	2.36	0.21	0.05	<LOD	1.58
Acenaphthylene	0.01	0.00	<LOD	0.20	0.04	0.01	<LOD	0.33	0.02	0.00	<LOD	0.14
Acenaphthene	<LOD	<LOD	<LOD	0.06	0.01	0.00	<LOD	0.09	0.01	0.00	<LOD	0.05
Fluorene	0.01	0.00	<LOD	0.21	0.01	0.00	<LOD	0.34	<LOD	<LOD	<LOD	0.06
Phenanthrene	0.03	0.01	<LOD	0.39	0.03	0.01	<LOD	0.85	0.01	0.00	<LOD	0.26
Anthracene	<LOD	<LOD	<LOD	0.29	<LOD	<LOD	<LOD	0.06	<LOD	<LOD	<LOD	0.03
Fluoranthene	0.01	0.00	<LOD	0.05	0.02	0.01	<LOD	0.10	0.01	0.00	<LOD	0.07
Pyrene	0.01	0.00	<LOD	0.05	0.03	0.00	0.01	0.09	0.01	0.00	<LOD	0.06
Retene	<LOD	<LOD	<LOD	0.03	0.01	0.00	0.01	0.05	0.01	0.00	<LOD	0.04
Benz[*a*]anthracene	<LOD	<LOD	<LOD	0.04	0.01	0.00	<LOD	0.04	<LOD	<LOD	<LOD	0.03
Chrysene	<LOD	<LOD	<LOD	0.04	0.01	0.00	<LOD	0.05	<LOD	<LOD	<LOD	0.02
Benzo[*b*]fluoranthene	<LOD	<LOD	<LOD	0.02	<LOD	<LOD	<LOD	0.02	<LOD	<LOD	<LOD	0.02
Benzo[*ghi*]perylene	<LOD	<LOD	<LOD	0.04	<LOD	<LOD	<LOD	0.04	<LOD	<LOD	<LOD	0.05
No Decontamination Control												
Total PAHs	0.44	0.12	0.04	2.92	0.43	0.09	<LOD	3.44	0.40	0.09	0.07	1.92
LMW PAHs	0.37	0.10	0.03	2.76	0.31	0.08	<LOD	3.23	0.25	0.07	0.03	1.78
HMW PAHs	0.04	0.01	0.00	0.27	0.08	0.01	<LOD	0.22	0.09	0.02	0.02	0.28
Naphthalene	0.20	0.05	<LOD	1.17	0.21	0.05	<LOD	1.93	0.11	0.04	<LOD	1.10
Acenaphthylene	0.01	0.00	<LOD	0.24	0.01	0.00	<LOD	0.18	0.02	0.00	<LOD	0.11
Acenaphthene	0.01	0.00	<LOD	0.07	0.01	0.00	<LOD	0.07	0.01	0.00	<LOD	0.05
Fluorene	0.01	0.00	<LOD	0.30	0.01	0.00	<LOD	0.24	0.01	0.00	<LOD	0.16
Phenanthrene	0.05	0.02	<LOD	1.04	0.04	0.01	<LOD	0.73	0.04	0.01	<LOD	0.35
Anthracene	<LOD	<LOD	<LOD	0.05	<LOD	<LOD	<LOD	0.06	<LOD	<LOD	<LOD	0.02
Fluoranthene	0.01	0.00	<LOD	0.10	0.02	0.01	<LOD	0.10	0.02	0.01	<LOD	0.10
Pyrene	0.01	0.00	<LOD	0.11	0.02	0.01	<LOD	0.10	0.03	0.01	<LOD	0.09
Retene	<LOD	<LOD	<LOD	0.06	0.01	0.00	<LOD	0.04	0.01	0.00	<LOD	0.04
Benz[*a*]anthracene	<LOD	<LOD	<LOD	0.10	<LOD	<LOD	<LOD	0.08	0.01	0.00	<LOD	0.13
Chrysene	<LOD	<LOD	<LOD	0.10	<LOD	<LOD	<LOD	0.02	<LOD	<LOD	<LOD	0.03
Benzo[*b*]fluoranthene	<LOD	<LOD	<LOD	0.02	0.01	0.00	<LOD	0.04	0.01	0.00	<LOD	0.02
Benzo[*ghi*]perylene	<LOD	<LOD	<LOD	0.02	<LOD	<LOD	<LOD	0.02	<LOD	<LOD	<LOD	0.02

GM: geometric mean; SE: standard error; LMW PAH: low molecular weight PAHs; HMW: high molecular weight PAHs; LOD: limit of detection

Table S4-4 Osmolality-corrected urinary PAH metabolite concentrations pre- and post-fire, and fold changes between pre- and post-fire levels. Values reported as µg per mmol solute. Bolded values indicate significant changes in post- versus pre-event fold-change values across all subjects; determined using a paired t-test on log-transformed values ($p<0.01$).

Inter-vention Group		**1-hydroxypyrene**			**Σhydroxy-phenanthrenes**			**Σhydroxyfluorenes**			**Σhydroxynaphthalenes**			**Total PAH metabolites**		
		Pre-fire	Post-fire	Fold Change	Pre-fire	Post-fire	Fold Change	Pre-fire	Post-fire	Fold Change	Pre-fire	Post-fire	Fold Change	Pre-fire	Post-fire	Fold Change
Wipe A	**N**	22	21	21	18	16	15	20	20	19	22	21	21	17	15	14
	GM	0.08	0.16	**1.96**	0.38	0.56	1.72	0.49	0.99	**2.13**	7.65	13.14	**1.69**	9.30	16.03	**1.86**
	Std Error	0.01	0.02	0.17	0.07	0.06	0.12	0.06	0.12	0.20	1.65	2.91	0.14	2.13	3.75	0.14
	Min	0.02	0.05	1.09	0.16	0.31	1.23	0.23	0.46	0.95	1.64	2.41	0.78	2.42	5.37	1.24
	Max	0.23	0.36	4.97	4.01	1.39	3.09	1.67	2.99	4.75	56.61	75.82	3.19	57.63	78.41	3.35
Wipe B	**N**	21	22	21	15	17	14	19	19	17	22	21	21	12	13	9
	GM	0.08	0.16	**1.95**	0.38	0.53	**1.45**	0.51	1.06	**2.12**	7.26	11.39	**1.63**	8.81	13.22	**1.81**
	Std Error	0.01	0.02	0.21	0.05	0.06	0.17	0.06	0.12	0.23	1.39	1.99	0.13	1.90	2.53	0.17
	Min	0.03	0.06	0.81	0.19	0.24	0.72	0.19	0.55	1.11	1.84	3.69	0.87	3.24	4.65	1.21
	Max	0.33	0.45	4.06	0.72	1.14	3.57	1.38	2.80	7.10	31.62	43.90	4.38	32.90	47.80	3.11
Detergent & Water	**N**	21	21	20	21	22	21	21	22	21	22	22	22	19	21	18
	GM	0.08	0.14	**1.94**	0.35	0.52	**1.53**	0.45	0.84	**1.87**	8.58	14.14	**1.65**	9.99	15.14	**1.71**
	Std Error	0.01	0.01	0.16	0.03	0.05	0.12	0.04	0.08	0.16	1.58	2.65	0.13	1.89	2.53	0.12
	Min	0.04	0.06	0.87	0.21	0.27	0.70	0.21	0.33	0.61	2.15	4.33	0.81	3.28	5.98	1.11
	Max	0.16	0.28	3.31	0.65	2.11	3.26	0.99	2.28	3.72	42.09	134.92	3.31	43.20	136.43	3.31
Control	**N**	22	22	22	19	19	17	21	22	21	22	22	22	18	19	16
	GM	0.07	0.14	**2.10**	0.28	0.45	**1.58**	0.43	0.79	**1.82**	8.02	13.04	**1.63**	9.70	15.91	**1.67**
	Std Error	0.01	0.01	0.20	0.02	0.05	0.12	0.04	0.06	0.19	1.46	2.16	0.13	1.90	2.73	0.13
	Min	0.03	0.06	0.87	0.17	0.17	0.78	0.23	0.32	0.56	2.22	4.25	0.76	3.93	5.49	1.16
	Max	0.15	0.43	5.47	0.52	1.35	2.77	1.42	1.63	4.71	81.75	105.03	3.69	82.69	106.50	3.36

Table S4-5 Creatinine-corrected urinary PAH metabolite concentrations by intervention, reported as ug/g creatinine. Bolded values indicate significant changes in post- versus pre-event fold changes; determined using a paired t-test on $\log_{10}$ transformed values ($p<0.01$).

		Wipe A					**Wipe B**					**Detergent & Water**					**Control**				
PAH metabolite		**N**	**GM**	**Std Err**	**Min**	**Max**	**N**	**GM**	**Std Err**	**Min**	**Max**	**N**	**GM**	**Std Err**	**Min**	**Max**	**N**	**GM**	**Std Err**	**Min**	**Max**
1-OH-pyrene	Pre-fire	22	0.05	0.01	0.01	0.20	21	0.05	0.01	0.01	0.17	21	0.04	0.01	0.01	0.12	22	0.04	0.01	0.01	0.18
	Post-fire	21	0.08	0.01	0.03	0.35	22	0.08	0.01	0.02	0.32	21	0.06	0.01	0.02	0.16	22	0.06	0.01	0.03	0.36
	Fold Change	21	**1.76**	0.22	0.63	7.26	21	**1.49**	0.22	0.42	4.68	20	**1.74**	0.23	0.65	11.01	22	**1.48**	0.23	0.26	4.89
ΣOH-phenanthrenes	Pre-fire	18	0.22	0.04	0.05	2.37	15	0.26	0.05	0.09	1.06	21	0.18	0.02	0.02	0.54	19	0.17	0.02	0.05	0.62
	Post-fire	16	0.27	0.04	0.14	1.53	17	0.29	0.04	0.09	1.03	22	0.23	0.03	0.10	0.75	19	0.20	0.03	0.08	0.89
	Fold Change	15	**1.47**	0.21	0.73	4.11	14	1.12	0.22	0.30	2.78	21	1.33	0.19	0.45	9.10	17	1.09	0.18	0.31	4.09
ΣOH-fluorenes	Pre-fire	20	0.28	0.04	0.10	1.07	19	0.34	0.05	0.08	1.13	21	0.23	0.03	0.03	0.74	21	0.27	0.04	0.06	1.01
	Post-fire	20	0.50	0.08	0.24	2.39	19	0.47	0.06	0.14	1.50	22	0.38	0.05	0.08	1.03	22	0.35	0.03	0.16	1.34
	Fold Change	19	**1.85**	0.27	0.57	6.03	17	1.41	0.24	0.43	4.88	21	**1.76**	0.25	0.40	8.80	21	1.26	0.21	0.27	5.74
ΣOH-naphthalenes	Pre-fire	22	4.35	1.01	0.60	47.10	22	4.64	0.99	0.81	56.03	22	4.35	0.76	1.51	33.03	22	4.97	1.02	1.07	96.52
	Post-fire	21	6.39	1.43	1.44	34.58	21	5.55	1.04	1.43	27.15	22	6.38	1.36	1.74	52.74	22	5.72	1.03	1.57	71.41
	Fold Change	21	**1.45**	0.19	0.53	6.75	21	1.25	0.16	0.33	3.45	22	**1.47**	0.16	0.52	7.34	22	1.15	0.18	0.25	6.52
Total PAH metabolites	Pre-fire	17	5.40	1.38	0.89	47.61	12	6.22	1.77	1.83	58.39	19	5.08	0.94	1.77	33.43	18	5.99	1.35	1.65	97.62
	Post-fire	15	7.87	1.94	3.16	38.84	13	6.26	1.35	1.87	29.56	21	6.81	1.33	2.31	53.33	19	6.87	1.32	2.02	72.02
	Fold Change	14	**1.58**	0.28	0.67	5.83	9	1.13	0.28	0.33	3.21	18	**1.60**	0.19	0.79	7.36	16	1.11	0.21	0.25	6.50

GM=geometric means.

References

IARC. 2002. Some traditional herbal medicines, some mycotoxins, naphthalene and styrene. IARC Monogr Eval Carcinog Risks Hum. 82:1–556.

IARC. 2010. Some non-heterocyclic polycyclic aromatic hydrocarbons and some related exposures. IARC Monogr Eval Carcinog Risks Hum. 92:1–853.

Silva J, Marques-da-Silva D, Lagoa R. 2021. Reassessment of the experimental skin permeability coefficients of polycyclic aromatic hydrocarbons and organophosphorus pesticides. Environ Toxicol Pharmacol. 86:103671, doi:10.1016/j.etap.2021.103671.

Chapter 5 Conclusions

Study outcomes

The three research chapters of this thesis demonstrate the prevalence of firefighters' exposures to carcinogenic and mutagenic combustion by-products, and the need for creative ways to reduce such exposures. Chapters two and three investigated firefighters' exposures to PAHs during emergency and training fire suppression; chapter four assessed the effectiveness of post-fire dermal decontamination to reduce internal dose of PAHs. Each chapter demonstrated that firefighters are occupationally exposed to combustion-derived carcinogens and mutagens, and that current protocols for decontamination do not appear to be effective.

The first research chapter was the first study of its kind to assess firefighters' exposures to combustion by-products during emergency fire and while on-shift suppression using a repeated measure designs (i.e., pre- and post-fire samples). The main outcomes of this chapter were as follows (1) airborne concentrations of lead and PAHs sometimes exceed occupational exposure limits, (2) firefighters experience significant increases of PAHs and metals on skin, clothing, and/or PPE from emergency fire suppression activities, (3) fire trucks have the most airborne contamination of PAHs and antimony, compared to offices and vehicle bays, but are still below occupational exposure limits, and (4) washing of PPE removes a majority of PAHs and measured metals.

Chapter three used a passive sampler approach to assess firefighters' exposures during live fire training. Passive samplers in the form of silicone wristbands (SWBs) were worn on the jacket and wrist of firefighters at training fire events. The main outcomes of this chapter were (1) current PPE protects firefighters from a majority of PAHs but not all, (2) Temperature, turbulence, and time of deployment likely limit the ability of SWBs to properly measure PAHs during live fire suppression activities, and (3) high molecular weight PAHs are most affected by temperature, turbulence, and time of deployment.

Finally, chapter four was an intervention study investigating the effectiveness of post-fire skin cleaning to reduce firefighters' internal dose of combustion emissions from live fire training. Several options are currently available for firefighters to clean their skin after fire suppression but none have been critically assessed until now. Three skin cleaning options were investigated: two commercially available skin cleaning wipes and a washcloth with dish detergent and water. Interventions were compared to the control (i.e., no skin cleaning). The results show that (1) firefighters are significantly exposed to PAHs from live fire training, (2) detergent and water is the most effective method at removing PAHs from skin after firefighting, but (3) none of the investigated skin cleaning interventions reduced the internal dose of PAHs that firefighters experienced.

Future Directions

Firefighters' exposures to PAHs during fire suppression were assessed in chapters two and three. A variety of proxies were used including air samples, wipes of surfaces (e.g., skin, clothing, PPE), SWs, and urine. Urine was the only biological sample collected due to logistical restraints. However, future work should explore alternative methods of assessing internal exposure and expanding the range of chemicals assessed. Urinary metabolites can be influenced by several other factors including the individual's diet, biological sex, participation in other activities, etc. Using a different proxy of internal PAH dose, such as saliva or blood, may generate additional data that is more immediate after a fire, thus leaving less room for uncertainties (e.g., other exposures prior to sample collection) and variability (e.g., interpersonal variations in metabolism), contributing the body of evidence that exposure comes from the specific fire suppression activity. Inter- and intrapersonal differences that may influence exposure (e.g., age, sex, ethnic origin, skin thickness, respiration rate, sweating), in addition to the phenomena that control exposure and internal dose (e.g., dermal contact, toxicokinetics), should also be further studied.

Chapter four assessed the effectiveness of post-fire skin decontamination. The results found soap and water to be the most effective at removing PAHs from the skin, compared to two commercially available skin wipes and a control (i.e., no skin decontamination). However, increases in internal dose of PAHs from the fire training event, as measured by urinary PAH metabolites, were the same between all intervention groups, suggesting that post-fire dermal decontamination did not reduce the amount of PAHs entering the body. In fact, penetration rates suggest that PAHs measured in urine are absorbed through the skin before skin decontamination would be possible (i.e., seconds). Future work should (1) assess the ability of post-fire dermal decontamination to reduce firefighters' exposures to, and cross contamination of, other combustion derived contaminants; (2) utilize computer models of dermal absorption and rates of absorption of combustion derived carcinogens and mutagens to determine if post-fire dermal decontamination is worth the cost for firefighters and (3) investigate other exposure prevention methods (i.e., rather than post-fire interventions) such as improved PPE.

Although research on firefighters' exposures has gained traction in the past decade, several populations of firefighters continue to be understudied. Women are often excluded due to limited numbers resulting in lack of statistical power, and non-municipal types of firefighting organizations are often excluded (e.g., remote, indigenous, military, and aviation) due to the convenience of sample collection from local, municipal firefighters. Furthermore, as climate change drives the rate and severity of wildfires, the need to investigate wildland firefighters' exposures is also high. Studies are limited due to the dynamic and extreme environments that wildland firefighters work in. Previous studies have assessed wildland firefighters' exposures, but they are often limited to prescribed burns and limited time points that are not always appropriate for the measured biomarkers of exposure. Creativity in study design and application of sampling techniques, that do not impede the firefighters' duties, are necessary to address these understudied populations.

Recent actions by the Canadian and American governments have provided the resources needed to aid firefighters in the years to come. In August 2021, the Canadian government announced an action plan to protect firefighters from toxic exposures experienced on the job including banning harmful flame retardants and assessing and monitoring levels of exposure (Government of Canada 2021). A few months later, the United States also announced a plan to help firefighters, more specifically addressing PFAS exposures including PFAS in firefighting foams, personal protective equipment, and other emergency response settings (The White House 2021). As research advances, and changes are made to reduce firefighters' exposures, biomonitoring of firefighter populations are necessary to ensure that changes are being effective.

The overarching goal is to reduce firefighters' disease incidence and mortality related to cancer and other illnesses. Firefighters' chemical exposures are just one piece of the puzzle. For firefighters and nonfirefighters alike, modifiable cancer risk factors, such as diet, smoking, exercise, and obesity, can influence one's cancer risk. In fact, one model suggested that excess body weight will become the second leading modifiable cause of cancer death, only after tobacco smoking, doubling cancer deaths of Canadians by 2047 (Pader et al. 2021). For firefighters specifically, studies have found several alarming statistics of modifiable risk factors including smoking rates of 13.6% and 17.4% for U.S. career and volunteer firefighters, respectively, obesity rates of nearly 80% of U.S. volunteer and career firefighters, and binge drinking behaviour in 58% of surveyed firefighters (Carey et al. 2011; Haddock et al. 2011; Poston et al. 2011). A less obvious cancer risk factor for firefighters is cortisol levels. Cortisol is a hormone that has been linked to cancer risk due to its immunosuppressive, obesogenic, and hyperglycaemic effects (Larsson et al. 2021). Cortisol levels can be affected by stress and disruptions in sleep, both of which have been demonstrated in firefighter populations (Lim et al. 2020; Perroni et al. 2009; Sopp et al. 2021). More specifically, Perroni et al. (2009) found a 109% increase in cortisol levels of firefighters after simulated firefighting, and Sopp et al. (2021) reported a significant positive correlation between cortisol levels and firefighters' PTSD symptoms (Perroni et al. 2009; Sopp et al. 2021). Relatedly, disruptions in circadian rhythm (e.g., for firefighters attending calls at night) can also influence cancer risk. Indeed, disruptions in circadian rhythm have been linked to increased rates of breast, ovarian, endometrial, prostate and hematological cancers, metabolic syndrome, and cardiovascular disease (Rana and Mahmood 2010; Shanmugam et al. 2014). In fact, the International Agency for Research on Cancer classified night shift work (i.e., work during the usual sleeping hours of the general population) as "probably carcinogenic to humans" (IARC 2020). Overall, to be effective, methods to reduce firefighters' cancer rates must address a broad range of issues, both personally and occupationally. More work is needed to address the many pieces that make up cancer risk for firefighters to create a comprehensive plan to reduce firefighters' risk. Even when ideal behaviours and protocols to reduce risk are established, it will be imperative to determine the most effective way to implement such changes. In discussions with firefighters worldwide, it is evident that cultural attitudes regarding exposure prevention and fire suppression tactics can differ significantly. Thus, communicating scientific results effectively, and working with fire departments to integrate meaningful changes, is of utmost importance. Furthermore, most research on firefighters' modifiable risk factors has come out of the U.S., leaving a significant literature gap when it comes to firefighters in the rest of the world and the aforementioned understudied groups more specifically. Examinations of understudied populations will permit evaluations of differences related to cultural and interpersonal factors.

Closing Statement

Firefighters face immediate and long-term health risks as part of their job. The results from my thesis demonstrated the significant exposures to combustion by-products that they experience during emergency and live fire training. The results also contributed to identifying the routes of exposure and ways to reduce exposures. It is imperative that we continue to work to establish evidence-based methods of reducing firefighters' chemical exposures and ultimately reduce their risk of cancer and other illness.

References

Carey, Mary G., Salah S. Al-Zaiti, Grace E. Dean, Loralee Sessanna, and Deborah S. Finnell. 2011. “Sleep Problems, Depression, Substance Use, Social Bonding, and Quality of Life in Professional Firefighters.” Journal of Occupational and Environmental Medicine / American College of Occupational and Environmental Medicine 53(8):928.

Government of Canada. 2021. “Government of Canada Announces Action Plan to Protect Firefighters from Harmful Chemicals.” Retrieved December 3, 2021 (https://www.canada.ca/en/environment-climate-change/news/2021/08/government-of-canada-announces-action-plan-to-protect-firefighters-from-harmful-chemicals.html).

Haddock, C. Keith, Nattinee Jitnarin, Walker S. C. Poston, Brianne Tuley, and Sara A. Jahnke. 2011. “Tobacco Use among Firefighters in the Central United States.” American Journal of Industrial Medicine 54(9):697–706.

IARC. 2020. “Night Shift Work.” IARC Monogr. Eval. Carcinog. Risks Hum. 124:1–371.

Larsson, Susanna C., Wei Hsuan Lee, Siddhartha Kar, Stephen Burgess, and Elias Allara. 2021. “Assessing the Role of Cortisol in Cancer: A Wide-Ranged Mendelian Randomisation Study.” British Journal of Cancer 2021 125:7 125(7):1025–29.

Lim, G., T. Jang, C. Sim, Y. Ahn, and K. Jeong. 2020. “Comparison of Cortisol Level by Shift Cycle in Korean Firefighters.” International Journal of Environmental Research and Public Health 17(13):1–14.

Pader, Joy, Yibing Ruan, Abbey E. Poirier, Keiko Asakawa, Chaohui Lu, Saima Memon, Anthony Miller, Stephen Walter, Paul J. Villeneuve, Will D. King, Karena D. Volesky, Leah Smith, Prithwish De, Christine M. Friedenreich, and Darren R. Brenner. 2021. “Estimates of Future Cancer Mortality Attributable to Modifiable Risk Factors in Canada.” Canadian Journal of Public Health 1–14.

Perroni, F., A. Tessitore, G. Cibelli, C. Lupo, E. D’Artibale, C. Cortis, L. Cignitti, M. De Rosas, and L. Capranica. 2009. “Effects of Simulated Firefighting on the Responses of Salivary Cortisol, Alpha-Amylase and Psychological Variables.” Ergonomics 52(4):484–91.

Poston, Walker S. C., C. Keith Haddock, Sara A. Jahnke, Nattinee Jitnarin, Brianne C. Tuley, and Stefanos N. Kales. 2011. “The Prevalence of Overweight, Obesity, and Substandard Fitness in a Population-Based Firefighter Cohort.” Journal of Occupational and Environmental Medicine 53(3):266–73.

Rana, Sobia and Saqib Mahmood. 2010. “Circadian Rhythm and Its Role in Malignancy.” Journal of Circadian Rhythms 8(1):1–13.

Shanmugam, Vignesh, Amro Wafi, Nawaf Al-Taweel, and Dietrich Büsselberg. 2014. “Disruption of Circadian Rhythm Increases the Risk of Cancer, Metabolic Syndrome and Cardiovascular Disease.” Journal of Local and Global Health Science 2013(1):3.

Sopp, M. R., T. Michael, J. Lass-Hennemann, S. Haim-Nachum, and M. J. J. Lommen. 2021. “Longitudinal Associations between Hair Cortisol, PTSD Symptoms, and Sleep Disturbances in a Sample of Firefighters with Duty-Related Trauma Exposure.” Psychoneuroendocrinology 134:105449.

The White House. 2021. “FACT SHEET: Biden-Harris Administration Launches Plan to Combat PFAS Pollution .” Retrieved December 3, 2021 (https://www.whitehouse.gov/briefing-room/statements-releases/2021/10/18/fact-sheet-biden-harris-administration-launches-plan-to-combat-pfas-pollution/).