Is Firefighting Carcinogenic?
Chemical Exposure and Cancer Risk Among Fire Fighters

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Introduction

Fire fighters inhale, ingest, and absorb hundreds of toxic, carcinogenic chemicals in smoke and soot during every phase of firefighting – suppression, knockdown/ventilation, and overhaul (clean-up). Moreover, with elevated body temperature during firefighting, skin absorption of chemicals can increase exponentially[1]. For every 5° increase in skin temperature, absorption increases 400% [1]. Fire fighter gear (helmets, uniforms, respirators) can be a source of ongoing exposure if not decontaminated after firefighting.

Fires today are more toxic than in the past because of the high usage of halogenated chemicals including brominated flame retardants (BFRs) in furniture, electronics, plastics, and other consumer products [2]. In the presence of high concentrations of bromine-containing materials, a major concern during fire events is the potential formation of large amounts of combustion by-products such as brominated dibenzo-p-dioxins and dibenzofurans (PBDD/Fs) [2-4]. PBDD/Fs are formed during fires under uncontrolled combustion conditions in the presence of chemical precursors such as the polybrominated diphenyl ethers (PBDEs) [3-5]. PBDD/Fs are major contaminants both indoors and in the environment [6], and contribute substantially to total dioxin-like toxicity in house dust [7], in food [8] and in human tissues [9,10].

When Flame-Retarded Furniture Burns…
The burning of flame-retarded foam furniture, plastics, and other treated household products releases large amounts of carcinogenic combustion by-products such as polychlorinated and polybrominated dibenzo-p-dioxins and dibenzofurans (PCDD/Fs, PBDD/Fs), mixed chloro-bromo dioxins and furans (PXDD/Fs).

Fire fighters have elevated rates of multiple cancers thought to be related to their occupational exposure to carcinogenic chemicals. In 2007, the International Agency for Research on Cancer classified firefighting as possibly carcinogenic to humans (Group 2B); relative risks were consistently increased for testicular cancer, prostate cancer, and non-Hodgkin's lymphoma[11,12]. A meta-analysis of 32 studies of fire fighters reported increased risks for these tumors as well as multiple myeloma[13]. A large-scale epidemiological study conducted by NIOSH, the National Cancer Institute, and the University of California-Davis reported higher risks of digestive, oral, respiratory, and urinary system cancers among 29,993 fire fighters in Chicago, Philadelphia, and San Francisco than among the general population[14]. A recent study[15] of 16,422 male Nordic fire fighters from five countries reported increased risk among fire fighters for prostate cancer and skin melanoma in fire fighters ages 40-49, and increased risk for multiple myeloma, adenocarcinoma of the lung, and mesothelioma in older fire fighters (70 years and above).

Overall, the data show that fire fighters are at increased risk for multiple cancers and their cancer risk increases significantly with the duration of firefighting.

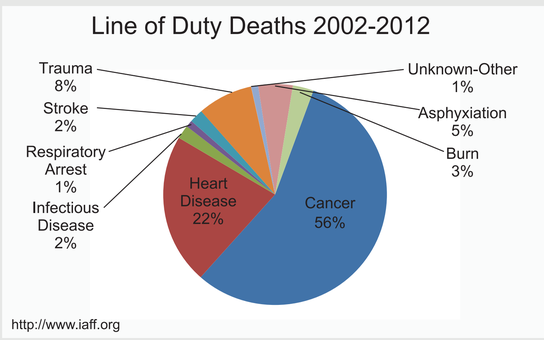

Figure 1. Cancer is the leading cause of line-of-duty deaths (LODDs) in the fire service today.

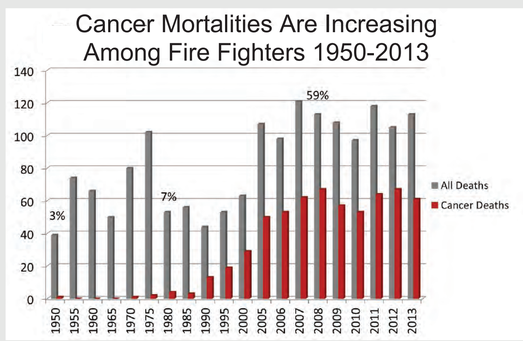

Figure 2. Since 1990, cancer-related mortalities among fire fighters have increased exponentially, accounting for 50-60% of all LODDs since 2005.

Exposure Assessment

Most investigations of fire fighter exposure have focused on a limited number of compounds (e.g., polychlorinated biphenyls, PCBs, PCDD/Fs) following acute fire events[16-20]. A recent pilot study of fire fighters from northern California represents the most extensive exposure assessment among fire fighters to date [21]. The California study was the first to analyze PBDD/Fs, along with PCDD/Fs, PBDEs, perfluorinated chemicals (PFCs), *p-p'*-DDE, hexachlorobenzene (HCB), tetrabromobisphenol-A (TBBPA) and bisphenol-A (BPA) in serum of fire fighters after a fire event. Based on the findings, a longitudinal study of chemical exposure and health outcomes in US fire fighters is planned.

The California Fire Fighter Study

The California cohort comprised 12 veteran San Francisco fire fighters including nine Caucasian males, two Asians (one male, one female) and one African-American male, ages 32 -59 years. Participants (1) had not worked in industries with known chemical emissions; (2) were fire fighters for at least 5 years; and (3) had responded to fire scenes at least 20 times in the past 5 years.

Congener-specific concentrations of PBDEs, PBDD/Fs, PCDD/Fs, and PCBs were determined in fire fighter serum samples by HRGC-HRMS; concentrations of PFCs, BPA, and TBBPA were determined by HPLC-MS/MS as previously described[21].

PBDEs

- PBDE levels in fire fighter serum were two to three times greater than average levels in the US population[22,23] and California residents[23].
- BDE-209 was the predominant congener, contributing, on average, 32% to the total PBDE content, followed by BDE-47 and -153. Given the short half-life of BDE-209 in human serum[24], this pattern implies ongoing exposure to DecaBDE during firefighting. Similarly, DecaBDE dominates the PBDE profiles in blood of e-waste recyclers engaged in open burning of plastic TVs and computers[25].

PBDD/Fs

- PBDD/F concentrations in fire fighter serum were similar to occupational levels in foam workers and 70-100 times higher than those in the general population (Table 1).
- Four detected PBDD/F congeners were present in two individuals: a 59-year old Caucasian male with the longest record of firefighting (28 years), who smoked and had hepatitis A, and a 40-year old Caucasian male who had been firefighting for 15 years without wearing personal protective equipment (PPE).
- PBDD/F dioxin-like toxicity (mean 103 pg/g lw WHO-TEQ) in fire fighter serum was 21 times higher than that of PCDD/Fs (5 pg/g lw WHO-TEQ), and 50-300 times higher than average population levels (Table 1), suggesting that exposure to PBDD/Fs may pose a significant health risk to individual fire fighters.

PCDD/Fs

- PCDD/F concentrations in fire fighter serum were relatively low (mean 5 pg/g lw WHO-TEQ); however, concentrations of 1,2,3,4,6,7,8-HpCDD exceeded levels of this congener reported in World Trade Center (WTC) responders[17]. HpCDD has been identified as a possible indicator congener in fire fighters[16].

PFCs

- PFOS was the prevalent PFC in fire fighter serum (mean 12 ng/ml wet weight, ww), followed by PFOA (7 ng/ml ww), and PFNA (2 ng/ml ww).
- PFOA and PFNA concentrations in California fire fighters were elevated relative to average population levels[26]. PFNA levels were also higher than those reported in World Trade Center fire fighters[20].

Other POPs

- *p-p'*-DDE and HCB in fire fighter serum were similar to average US population levels[27] and PCB congener profiles were not indicative of occupational exposure.
- TBBPA was not detected; BPA was detected at trace levels in fire fighter serum.

Table 1. Comparison of PBDD/F Concentrations (pg/g lw) in Fire Fighters, Foam Workers, and General Population

Population	Location	Year	Tissue	2378-TBDD	2378-TBDF	12378-PeBDF	23478-PeBDF	ΣPBDD/F	TEQ	Ref
Fire fighters	California	2009	serum	58 (nd-356)	42 (nd-504)	126 (nd-922)	126 (nd-996)	352	103[c]	21
Extruder operators	Germany	1990-91	blood	40 (nd-478)	8 (nd-112)				41[c]	28[a]
General pop.	Sweden	2007	adipose	nd	0.7 (0.3-2.2)	0.1 (nd-0.9)	0.1 (nd-0.5)	1 / 2.3	0.5/0.6[d]	9[b]
General pop.	Japan	1970	adipose	1.7 (nd-4.2)	3.3 (1.6-4.3)		0.3 (0.3-0.6)	5.1	2.1[c]	29
General pop.	Japan	2000	adipose	0.51 (0.1-2)	2.8 (1.7-4.2)		1 (nd-1.9)	3.4	1.1[c]	29
General pop.	Various		milk	(0.1-0.3)	0.7 (nd-2.7)		0.2 (nd-1.1)		0.3/1.1[e]	10

[a] Data from foam workers in a plant that does extrusion and blending of polybutyleneterephthalate with DecaBDE
[b] Lower bound (excluding <= values)/ Upper bound (including <= values)
[c] TEQ calculated with PCDD/F WHO_{2005} TEFs[30]; [d] TEQ calculated with PBDD/F REPs[31];
[e] TEQ calculated with PCDD/F WHO_{1998} TEFs[32]; see discussion of TEQ calculations in 21.
nd= not detected

Conclusions

- Overall the data indicate that occupational exposure to toxic, carcinogenic chemicals including flame retardants and combustion by-products (PBDD/Fs) places fire fighters at increased risk for cancer, stroke, and other serious health effects.
- The California study showed that fire fighters accumulate high levels of PBDEs, PBDD/Fs, and PFCs in blood while firefighting, and PBDD/Fs may contribute substantially to dioxin-like toxicity in individual firefighters.
- The predominance of of DecaBDE (BDE-209) in fire fighter blood suggests continuous exposure to DecaBDE released from burning plastics.
- Elevated PFOA and PFNA concentrations in fire fighter blood implies that firefighting involves substantial exposure to long-chain PFCs currently used as replacements for banned PFCs.
- A longitudinal study is planned to examine chemical exposure and biomarkers of health effects including cancer in fire fighters across the country.

References

1. Firefighter Cancer Support Network (2013) http://www. firefightercancersupport.org. 2. Shaw SD, et al. (2010) *Rev. Environ. Health* 25: 261–305. 3. Ebert J, Bahadir M. (2003) *Environ. Int.* 29: 711–716. 4. United Nations Environment Program (UNEP) (2010) http://www.chm.pops.int/. 5. Kannan K, et al. (2012) Dioxins and Health, John Wiley and Sons, Inc., New York, pp 255–302. 6. Birnbaum LS, et al. (2003) *Environ. Int.* 29: 855-860. 7. Suzuki G, et al. (2010) Fifth International Symposium on Brominated Flame Retardants, Kyoto, Japan. 8. Rose M, et al. (2010) *Organohalogen Compds* 72: 1396 - 1401. 9. Jogsten IE, et al. (2010) *Chemosphere* 78: 113–120. 10. Kotz A, et al. (2005) *Organohalogen Compds* 67: 1540-1544. 11. International Agency for Research on Cancer (IARC). (2010). Vol. 98. http://www.monographs.iarc.fr/. 12. Straif K, et al. (2007) *The Lancet Oncology* 8: 1065-1066. 13. LeMasters GK, et al. (2006) *J. Occup. Environ. Med.* 48: 1189-1202. 14. Daniels RD, et al. (2013) *Occup. Environ. Med.* Online: 14 Oct 2013. doi:10.1136/oemed-2013-101662. 15. Pukkala E, et al. (2014) *Occup. Environ. Med.* Online: 6 Feb. 2014: doi: 10.1136/oemed-2013-101803. 16. Edelman P, et al. (2003) *Environ. Health Persp.* 111: 1906–1911. 17. Horii Y, et al. (2010) *Environ. Sci. Technol.* 44: 5188-5194. 18. Schecter A, et al. (2002) *Chemosphere* 47: 147-156. 19. Hsu J-F, et al. (2011) *Chemosphere* 83: 1353-1359. 20. Tao L, et al. (2008) *Environ. Sci. Technol.* 42: 3472–3478. 21. Shaw SD, et al. (2013) *Chemosphere* 91: 1386-1394. 22. Schecter A, et al. (2005) *J. Occup. Environ. Med.* 47: 199-211. 23. Zota AR, et al. (2008) *Environ. Sci. Technol.* 42: 8158-8164. 24. Thuresson K, et al. (2006) *Environ. Health Persp.* 114: 176-181. 25. Bi X, et al. (2007) *Environ. Sci. Technol.* 41: 5647–5653. 26. Calafat AM, et al. (2007) *Environ. health Persp.* 115:5647-5653. 27. Patterson Jr DG, et al. (2009) *Environ. Sci. Technol.* 43: 1211-1218. 28. Zober MA, et al. (1992) *Br. J. Ind. Med.* 49: 532-544. 29. Choi J-W, et al. (2003) *Environ. Sci. Technol.* 37:817-821. 30. Van den Berg M, et al. (2006) *Toxicol. Sci.* 93: 223–241. 31. Samara F, et al. (2009) *Environ. Int.* 35: 588–593. 32. Van den Berg M, et al. (1998) *Environ. Health Perspect.* 106: 775–792.

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