Haze Disaster in South East Asia: An Urgent Study on the Effect of Dioxins to the Firefighters

In 2015, the National Institute for Occupational Safety and Health (NIOSH) reported that fire fighters from Chicago, Philadelphia, and San Francisco showed higher rates of certain cancers (cancer of digestive, oral, respiratory, urinary, bladder, prostate and malignant mesothelioma) than the general population [13]. Elevated rates of cancer, including four types that are potentially related to exposure to PCDD/Fs—multiple myeloma, non-Hodgkin’s lymphoma, prostate, and testicular cancer in firefighters have been reported in several studies [14-16]. Occupational exposure to PCDD/Fs in firefighters has also been investigated by Tsai et al. [17], Chernyak et al. [18] and Hsu et al. [19].

In Malaysia, firefighters might be exposed to additional hazard during the forest fire or when the haze disaster occurs in neighbouring country. According to the New York Times [20], the experts in public health and atmospheric modeling from Harvard and Columbia estimated that 91,600 people in Indonesia, 6,500 in Malaysia and 2,200 in Singapore may have died prematurely because of exposure to fine particle pollution known as PM 2.5 from burning forests, in particular carbon-rich peatlands. Based on our preliminary study and the data from the Fire and Rescue Department of Malaysia, no death is reported in the haze incident. However, the effect of the haze is expected to be more severe especially for the firefighters who are at the front line spending hours in burning scene than the normal population. This urges an immediate study on the occurrence and level of dioxins and dioxins-like compounds among the firefighters.

Exposure to environmental contaminants can be prevented. Raised awareness and exposure prevention efforts are cost-effective means to reduce occupational cancer risk. Thus, the fire service should increase efforts to educate their members about the safe work practice that includes proper training, proper use of protective clothing or apparatus, and proper use of approved respiratory protection during all phases of firefighting. Incentive should also be given to the industries and individuals who involve in the innovation and invention of effective and advance protective gears.

Editorial

Firefighters may be exposed to a wide variety of toxic chemicals in their line of work, including volatile organic compounds, polycyclic aromatic hydrocarbons (PAHs), polychlorinated biphenyls (PCBs), brominated flame retardants (BFRs), metals, and various combustion by-products [1-4]. Such exposures can occur through inhalation and skin contact, although advances in personal protection (e.g. clothing and breathing apparatus) have apparently reduced such risks in recent years. However, the major concern during fires is the potential formation of large amounts of harmful by-products such as chlorinated and brominated dibenzo-p-dioxins and dibenzofurans (PCDD/Fs and PBDD/Fs) [5,6] and polychlorinated biphenyls (PCBs) [7]. All of these contaminants belong to the persistent organic pollutants (POPs) and highly toxic substances that cause adverse effects on humans and ecosystem. Their toxic responses include immunotoxicity, carcinogenicity and several endocrine effects related to reproduction [8].

With the modern lifestyle, consumer goods are increasingly manufactured using synthetic materials, and the use of various kinds of electronic gadgets or devices create more smokes and toxic substances when catch fire. According to Zennegg et al. [9], the open burning of electronic–waste containing polybrominated diphenyl ethers (PBDEs) is estimated to release tons of PBDD/Fs and PCDD/Fs into the environment. In the United Kingdom, PBDD/Fs and PCDD/Fs contribute to about 30% of the dioxin–like toxicity in food [10]. A recent study of Swedish adipose tissue samples demonstrated that PBDD/Fs may contribute up to 14% of the total dioxin toxic equivalents (TEQs) [11]. Similarly, Kotz et al. [12], found that the TEQ of PBDD/Fs may account for up to 12% of the dioxin-like toxicity in human milk.

References


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