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PM_{10}-bound polycyclic aromatic hydrocarbons: Biological indicators, lung cancer risk of realistic receptors and 'source-exposure-effect relationship' under different source scenarios

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ABSTRACT

Cancer has become a critical health issue in the world heritage city Kandy, Sri Lanka. Polycyclic aromatic hydrocarbons (PAHs), one of persistent organic pollutants, in the atmosphere may be a major etiological factor in lung carcinogenicity. Over the very high concentrations of ambient air PAHs reported in Kandy, this paper is focused on setting priorities to control human exposure to PAHs in prevention of cancer.

On re-appraisal of the classical indicator benzo(a)pyrene (B[a]P) for atmospheric PAHs-related carcinogenicity, B[a]P failed to reflect the toxicity completely and may not be the sole indicator for risk assessment studies in complex multi-sourced urban environments. The excess lifetime lung cancer risks of atmospheric PAHs with 'less than lifetime exposure' were assessed based on both 'B[a]P toxic equivalents' and 'B[a]P surrogate epidemiological' approach of risk quantification, over emissions characterized urban, suburban, and rural areas of Kandy. In urban heavy traffic areas, PAH-related additional cancer burden has been 942 million^{-1} over 30 y of exposure. Over the whole study area, $\sum$p-PAHs show strong correlation ($r = 0.8$) to the predicted risk levels. While the urban and suburban predicted cancer risk levels could not show significant correlation to their emission sources indicating the real complexity in mega urban environments, the rural lung cancer risk levels correlated perfectly with the source, firewood combustion.

Policy decisions on environment and health could be based on established correlations among 'emission sources-exposures-health effects'. The priority for ''analysis of options and policy formulation to reduce inhalation PAHs exposure of population in Kandy'' was considered ''moderate to high''.

1. Introduction

Polycyclic aromatic hydrocarbons (PAHs), a group of potent carcinogenic and genotoxic agents, with long-term evidence were suggested to elevate the risk of various cancers. Most of the data collected on the human health effects of PAH exposure arise from occupational epidemiological studies; but such data on environmental exposure are deficient due to the complexity of the issue. Numerous studies on coke oven workers have provided evidence of a dose–response relationship between many PAHs and health effects, including lung cancer and depressed immune function (WHO/IPCS, 1998). However, lung cancer has been the critical endpoint for health risk evaluation due to the well-documented carcinogenicity of several PAHs (IARC, 1987). The bulk of information on toxicity and occurrence of PAHs is related to benzo(a)pyrene (B[a]P), which is by far the most intensively studied PAH in experimental animals and reported to develop lung tumors in hamsters upon inhalation (Thyssen et al., 1981). Certain animal experiments have shown that the carcinogenic activity of PAHs in vehicle exhaust extracts is associated mainly with the fraction containing compounds composed of 4–7 aromatic rings (Grimmer et al., 1984).

Cancer is a leading cause of death worldwide, accounting for 7.6 million deaths (~13% of all deaths) in 2008, and projected to grow over 11 million in 2030 (WHO, 2011). Among all cancers worldwide, combined in both sexes the highest rate of incidence (23%) and mortality (19.4%) were reported with lung cancer by 2008 (Globocan, 2008). In Sri Lanka, neoplasm has been identified as the second leading cause of hospital deaths in the country (with a mortality rate of 17.5/100000 population and a proportionate mortality of 10.1) and in Kandy district (Annual Health Statistics of Sri Lanka-2007). Further, the available most recent statistics of National Cancer Institute (2007) revealed that, among the new male patients registered, carcinoma of bronchus and lung has been the foremost site. They all were indicative of the status of

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neoplasm and the trend for lung cancer in Sri Lanka. However, >30% of cancer deaths can be prevented by avoiding or modifying key risk factors (WHO, 2011) including urban air pollution, that is attributed mainly to vehicular and other stationary emissions. One of the biggest challenges faced by cities in planning the city-specific policy programs for prevention and control of cancer is the identification of local cancer risk factors associated with sources of emission, especially inhalable carcinogens.

In urban environments with relatively high population and traffic volume and/or density, human exposure to hazardous substances is of greater possibility; which may be compounded by the urban topography and microclimate that contribute to poor dispersion conditions. As PAHs are formed during incomplete combustion or pyrolysis of organic material, they are ubiquitous and unavoidable contaminants especially in the urban environment. Therefore, in prevention of PAH induced cancer; selection of biological indicators for risk quantification, derivation of limit values for continuous ambient air monitoring, emission control in order to reduce human exposure and health impact assessment of inhalational PAH exposure to build 'environmental dose–response relationship' are timely and of high importance. While no specific guideline value set by WHO for ambient air PAHs, it specifies a risk estimate as a basis for policy makers (WHO, 1987, 2000). On the present knowledge, B[a]P has been the most appropriate indicator for the carcinogenic fraction of the large number of PAHs in air although the limitations and uncertainties in such an approach were recognized (WHO, 2000).

Concerning PAHs, for the first time in Sri Lanka, an extensive study was carried out based on the hill capital Kandy. It revealed very high outdoor air concentrations compared to other reported cities in the world, and vehicular emissions and firewood combustion were identified as the attributed sources (Wickramasinghe et al., 2011). Being the initiative of a long-term programme directed towards the final objective of reducing the lung cancer risk from short term, long term and life time exposure to atmospheric PAHs in Kandy, this study could provide information with wide applicability and can be used by similar cities in Sri Lanka and anywhere in the world too.

One of the purposes of this paper is therefore, to evaluate for the biological indicators of ambient air PAHs in general and also specifically under different sources of emission in Kandy, with re-appraisal on occupationally based classical indicator B[a]P. Secondly, it assesses how the excess lifetime lung cancer risk (ELTLCR) of atmospheric PAHs with 'less than lifetime exposure' of different types of realistic receptors varies relative to that from generally practiced 'continuous lifetime exposure'. It was based on both 'B[a]P toxic equivalents approach' and 'B[a]P surrogate epidemiological approach' of risk quantification (WHO/IPCS, 1998), using the measured ambient air particulate PAH concentrations distributed spatially over urban, suburban, and rural areas of Kandy. Thirdly, the correlations among emission sources, human exposure and screening level health risks were established. Those correlations together with obtained risk estimates, as an evidence-based strategy, would have very important applications in the overall mechanism of reducing the burden of cancer by exposure alleviation. Further, it reveals the parameters which can be manipulated for exposure reduction especially over the identified high-risk areas/community groups; all in turn can be used in implementing community health programmes, environmental policy formulations and related urban planning. Furthermore, while the reported epidemiological data on the ambient air PAH exposures are still scarce, the risk estimates presented in this work may be useful in taking into consideration in the design of the framework for the future confirmatory epidemiological studies on lung cancer, possibly assisted by surveillance and descriptive studies, in Kandy.

2. Materials and methods

2.1. Outdoor air sampling and analytical method

The study was conducted in the municipal region of Kandy, Sri Lanka, over randomly selected twenty sampling sites as to be representative of the stratified whole study area for accurate and generalized exposure data of the targeted population. All the details on site description and classification, air sampling and analytical procedures have been described in an already published article (Wickramasinghe et al., 2011), and only a brief account is given here. All sampling stations were basically classified into three major categories: '*Urban-Commercialized areas*' [U-C],8 sites; '*Suburban-Mixed areas*' [S-M], 7 sites; '*Rural-Residential areas*' [R-R], 5 sites, and were further classified on the type of possible main sources of emission' as '*Traffic*' [T] or '*Background stations*' [B]. The heavy traffic [HT] stations (>15 000 vehicles d^{-1}) in [U-C] and [S-M] areas were sub-defined as Urban Heavy Traffic [U/HT] and Suburban Heavy Traffic [S/HT]; similarly the light traffic [LT] stations (>2000 < 15 000 vehicles d^{-1}) were termed as [U/LT] and [S/LT]. The sampling sites in the [R-R] area were all background stations and were further sub defined primarily on quantitative firewood use; Rural Background-High Firewood [R/B/HF] (>50 kg d^{-1}) and Rural Background-Low Firewood [R/B/LOF] (15–50 kg d^{-1}).

Eight hours (from 9 a.m.) samplings for PM_{10} bound PAHs in ambient air were performed with a high volume Respirable Dust Sampler (Envirotech Model APM 460) with glass micro fiber filter paper (Whatman EPM 2000; 20.3 × 25.4 cm), from July/2008 to March/2009. Samples were analyzed for the US EPA priority 16 PAHs according to the modified and optimized procedure developed by Wickramasinghe and Karunaratne (2008) based on US EPA Compendium Method TO-13A (US EPA, 1999).

2.2. Cancer potency and contribution to total carcinogenicity

Since no suitable data available from human studies for assessing the potency of individual PAHs, the potency of B[a]P and other PAH species in humans is estimated using studies in animal models, assuming that the relative potency of two PAHs in an animal model is similar to that of the same compounds in humans (Albert et al., 1983; Nesnow, 1990). There is a reasonable degree of agreement between the relative potency values of individual PAH compounds relative to B[a]P, referred as estimates of toxicity equivalence factors (TEFs), calculated by different toxicity equivalence models for the carcinogenic PAHs (WHO/IPCS, 1998). Among those, the TEF scheme laid by Larsen and Larsen (1998) was used in the current study (Table 1), because it has been based on the extensive database on carcinogenic studies using various routes of administration. The TEF values are quite similar to other schemes, such as those of Nisbet and Lagoy (1992), but the TEF for fluoranthene (Fl) is 0.05 compared with 0.001, which makes the difference. Fluoranthene, being an experimental mutagen with abundance in emissions from combustion (WHO/IPCS, 1998), the given higher TEF value may better describe its contribution to the risk from PAHs exposure. By multiplying the concentration of individual PAHs with their TEF values, the carcinogenic activity were calculated as B[a]P equivalents (WHO/IPCS, 1998) for different localities. Developed in early 1990s, the TEF approach is still the most widely used method in PAH related toxicological studies (see e.g. Liu et al., 2010; Callén et al., 2011; Tom et al., 2011).

2.3. Exposure concentration (EC) based on the duration and the pattern of exposure

The probabilistic risk assessment (PRA) was not adopted in the current study as conventional, because it is limited by the