Health risk assessment for residents exposed to atmospheric diesel exhaust particles in southern region of Taiwan

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HIGHLIGHTS

- DEP dose and cancer risk estimates showed heterogeneously spatiotemporal difference.
- DNA damage biomarker and cancer incidence estimates had a positive association.
- Resident health risk from atmospheric DEP depending on measured data type.
- Health risk assessments of air pollution can guide adaptive mitigation strategies.

ARTICLE INFO

ABSTRACT

Evidence shows a strong association among air pollution, oxidative stress (OS), deoxyribonucleic acid (DNA) damage, and diseases. Recent studies indicated that the aging, human neurodegenerative diseases and cancers resulted from mitochondrial dysfunction and OS. The purpose of this study is to provide a probabilistic risk assessment model to quantify the atmospheric diesel exhaust particles (DEP)-induced pre-cancer biomarker response and cancer incidence risk for residents in south Taiwan. We conducted entirely monthly particulate matter sampling data at five sites in Kaohsiung of south Taiwan in the period 2002–2003. Three findings were found: (i) the DEP dose estimates and cancer risk quantification had heterogeneously spatiotemporal difference in south Taiwan, (ii) the pre-cancer DNA damage biomarker and cancer incidence estimates had a positive yet insignificant association, and (iii) all the estimates of cancer incidence in south Taiwan populations fell within and slight lower than the values from previous cancer epidemiological investigations. In this study, we successfully assessed the tumor incidence for residents posed by DEP exposure in south Taiwan compared with the epidemiological approach. Our approach provides a unique way for assessing human health risk for residences exposed to atmospheric DEP depending on specific combinations of local and regional conditions. Our work implicates the importance of incorporating both environmental and health risk impacts into models of air pollution exposure to guide adaptive mitigation strategies.

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1. Introduction

Growing evidence shows that there were significant associations among air pollution, oxidative stress, deoxyribonucleic acid (DNA) damage, and diseases (Klaunig and Kamendulis, 2004; Demirbag et al., 2005; Altindag et al., 2007). Lin and Beal (2006) indicated that mitochondrial dysfunction (e.g., DNA damage) and

oxidative stress (e.g., production of reactive oxygen species (ROS)) were highly likely to pose aging related and human neurodegenerative diseases including Alzheimer's disease (AD), Parkinson's disease (PD), Amyotrophic lateral sclerosis (ALS), and Huntington's diseases (HD). Thus, many aging related diseases, including cancer (Klaunig and Kamendulis, 2004), coronary artery disease (CAD) (Demirbag et al., 2005), human neurodegenerative diseases (AD, PD, ALS, and HD) (Lin and Beal, 2006), and rheumatoid arthritis (RA) (Altindag et al., 2007), were caused by DNA damage through ROS production and accumulation.

Kappos et al. (2004) indicated that per 10 μg m^{-3} PM$_{2.5}$ (particulate matter (PM) with aerodynamic diameter ≤ 2.5 μm)

increase resulted in mortality up to 13–14% (95% CI: 4.2–23%) for all causes, 18–19% (95% CI: 5.8–33%) for cardiopulmonary disease, and 18–20% (95% CI: −8.4 to 60%) for lung cancer. Krewski et al. (2000) and Pope et al. (2002), however, indicated that the estimated mortality was 4.1–7.0% (95% CI: 0.8–11%) for all causes, 5.9–12% (95% CI: 1.5–17%) for cardiopulmonary disease, and 0.8–13.5% (95% CI: −8.7 to 23%) for lung cancer per 10 μg m^{-3} $PM_{2.5}$ increase. Several studies have performed the DNA damages by using several biomarkers, such as determinations of 1-hydroxypyrene (1-OHP), 8-hydroxydeoxyguanosine (8-OHdG or 8-oxodG), disease activity, DNA adducts and formamidopyrimidine glycosylase (FPG), through detecting tissues, plasma, and urine (Chuang et al., 2003; Altindag et al., 2007).

Here we focused on a highly industrialized (more than 60% of Taiwan heavy industries) and densely populated area, Kaohsiung, located in south Taiwan (Yuan et al., 2002; Wang et al., 2006). Kaohsiung is the second largest metropolitan area in Taiwan. Moreover, Kaohsiung is a densely populated region (nearly 2.78 million persons within a total area of 3000 km^2). Yuan et al. (2002) reported that the neighborhood of Kaohsiung has the worst ambient air quality in that the haze days of the Pollution Standard Index (PSI) greater than 100 was about 9–10% during 1999–2000. Several epidemiological studies have also evaluated the adverse health outcomes exposed to air pollutants for susceptible populations in south Taiwan (Chen et al., 1998; Cheng et al., 2006).

In the recent year, the chemical compositions, source identification, and their environmental impacts (e.g., visibility) of atmospheric aerosols were concerned inseparably (Yuan et al., 2002; Tsai and Chen, 2006a; Wang et al., 2006). The epidemiological studies of the human health outcomes have investigated, especially for cancer incidence among occupational and non-occupational populations. However, the issue of the adverse health effects (e.g., asthma, respiratory diseases, tumor incidence) caused directly (not epidemiological study) from atmospheric aerosols in the area was rarely addressed (Wang et al., 2006).

The most effective way to study the impacts of atmospheric air pollutant on human health is through a mechanistic modeling because it resolves some of the limitations associated with empirically based statistical techniques. These limitations include the lack of long-term and continuous air pollutant data. Because of a scientific consensus that air pollutants are occurring with associated human health consequences, public health research has focused on identifying and implementing effective mitigation and adaptive strategies.

The purpose of this study is to provide a probabilistic risk assessment model to quantify the atmospheric diesel exhaust particles (DEP)-induced pre-cancer biomarker response and cancer incidence risk for residents in south Taiwan. It is recognized that one of challenges for public health responses to air pollutants is the need for location-specific risk assessment. This study addresses this challenge by providing a unique way for assessing human health risk for residences posed by atmospheric DEP depending on specific combinations of local and regional conditions.

2. Materials and methods

2.1. Data sources

There are five selected sampling sites that are all located in Kaohsiung: (i) Meinung (Site M), (ii) Chiautou (Site C), (iii) Jenwu (Site J), (iv) Daliao (Site D), and (v) Linyuan (Site L). Several industrial and agricultural mixed areas are scattered in this study area (Fig. 1A). Fig. 1B gives the locations shown with the Universal Transverse Mercator (UTM) Grid System along with population

densities. Among the five sites, Site M is considered as a less polluted small town with lower industrial and vehicle emissions compared to the other sites. The other four sites (Sites C, J, D, and L), however, are close to many industrial complexes with petrochemical and metal manufacturing plants in south Taiwan. Therefore, there were 60% of Taiwan's petrochemical plants and over 5000 factories located in this study area, emitting nearly 39.6 ktons of TSP and 17.4 ktons of PM_{10} in 1997 (CTCI Corporation, 1999).

All PM_{10} and $PM_{2.5}$ samples at each site were collected using personal environmental monitor (PEM, MSP corp.) and Harvard samplers (Air Diagnostics and Engineering) on a 24-h basis placed 3 m apart on the roof of a building about 10–15 m above ground. The air flow rate of PEM was settled at 10.0 ± 0.11 pm and used 37 mm diameter quartz (Pallflex 2500 QAT-UP) filter paper as sampling media. The sampling period covered September 2002–August 2003 with autumn (September–November), winter (December–February), spring (March–May), and summer (June–August) seasons. All 110 samples were collected for chemical and statistical analyses. The statistical analysis was based on chemical mass balance (CMB) modeling (Watson et al., 1991).

After sampling, each sample was weighed by an analytical balance (Mettler, Toledo AT261), and then water soluble ions (including F$^-$, Cl$^-$, NO$_3^-$, SO$_4^{2-}$, Na$^+$, NH$_4^+$, K$^+$, Mg^{2+}, and Ca^{2+}), carbonaceous contents (including organic carbon (OC) and elemental carbon (EC)), and metals (including Ag, Al, As, Ba, Ca, Cd, Co, Cr, Cs, Cu, Fe, Hg, K, Mg, Mn, Na, Ni, Pb, Rb, Se, Sr, Ti, Tl, V, and Zn) were analyzed by using Dionex DX-120 Ion Chromatograph, Heraeus CHN-O-Rapid elemental analyzer, and Agilent Model 7500 Inductively coupled plasma mass spectrometry, respectively (Tsai and Chen, 2006a; Wang et al., 2006). Details of the sampling program, chemical analysis protocols, and CMB modeling have been described in previously studies (Lai et al., 2003; Wang et al., 2006). Briefly, the CMB source apportionment technology can be described as follow equation

$$X_i = \sum_{j=1}^{J} F_{ij}S_j,\tag{1}$$

where X_i is the concentration of element i, F_{ij} is the fraction of element i in source j, S_j is the contribution of source j (Watson et al., 1991). Here, our observed data was collected with at least 2 consequence sampling days per month each site, except for August. Therefore, the sample size for each site was 22.

2.2. Problem formulation

Here we used the source apportionment technology to estimate the DEP contributory ratio from published $PM_{2.5}$ data measured in selected five sites in south Taiwan (Lai et al., 2003). Residents lived in the study area were more concerned, especially for elderly subgroup. Fortunately, we had enough information on monthly, seasonal, and annual $PM_{2.5}$ to estimate the human adverse effect of tumor incidence. We employed Crystal Ball® (Version 2000.2, Decisioneering, Inc., Denver, Colorado, USA) to perform the Monte Carlo (MC) simulation. All of the simulation frequencies were 10,000 iterations to ensure the stability of results. Lognormal distributions were fitted to the measured $PM_{2.5}$ data by maximizing the log-likelihood function.

2.3. Exposure assessment

Our exposure assessment model based on a monthly-basis vehicle contribution can be expressed as