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Research Article

Health Risk of Ambient $PM_{2.5}$ Concentration: A Case study of New Delhi, India

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A B S T R A C T

New Delhi, the national capital city of India ranks among the world's most polluted cities according to World Health Organization (WHO). This megacity suffers from alarming levels of $PM_{2.5}$, a major threat to human health. The prolonged exposure of fine particulate matter resulted into the various life threatening diseases such as lung cancer, respiratory and tuberculosis, asthma, Chronic Obstructive Pulmonary Disease (COPD) etc. In 2009, the Central Pollution Control Board (CPCB) found it beneficial to implement some revised control strategies on emissions via National Ambient Air Quality Standard (NAAQS), originally implemented in 1981. The threshold of the prescribed values for fine particulate matter ($PM_{2.5}$) have been set at 60 µg/m^3 for a 24-hour period and 40 µg/m^3 for an annual period. Both are considered safe concentration limits regarding human health effects. However, the daily and annually averaged concentrations of $PM_{2.5}$ have been observed to be three to four fold of NAAQS in the present study of the year 2012. This study relates the incessant exposure of $PM_{2.5}$ to deleterious health effects, such as heart and lung diseases. The relation between environmental air quality and human health has been analyzed through different parameters e.g., Hazard Quotient (HQ) and Hazard Index (HI) in New Delhi. HQ for inhalation corresponding to average concentration is 3.98 and 2.412 at IGI airport and ITO respectively. Consequently HI corresponding to average concentration of $PM_{2.5}$ is 3.981 (> 1) and 2.413 (> 1) at IGI airport and ITO respectively, indicates high health risk due to $PM_{2.5}$ exposure.

Keywords: $PM_{2.5,}$ $PM_{10,}$ Hazard Quotient, Hazard Index, Health Effect

Introduction

Air pollution is one of the major causes of concern, particularly in developing countries such as India, Bangladesh and China. Currently, in India, especially in Delhi, air pollution is widespread in urban areas. Vehicles are the major contributors while industries and thermal power plants also play significant role (Srivastava and Jain 2003). According to the findings of report, Global Burden of Disease (GBD, 2013) after high blood pressure, indoor air pollution, smoking and poor nutrition, the fifth largest killer in India is air pollution. In India, each year there are approximately 6,20,000 premature deaths due to the diseases related to air pollution only, which was 1,00,000 in the year 2000, a six-fold increase.

It is being recognized as a major threat to the human health. The United Nations Environment Programme has estimated that globally 1.1 billion people breathe unhealthy air (UNEP, 2002). Epidemiological studies have shown that

concentrations of ambient air particles are associated with a wide range of effects on human health, especially on the cardio-respiratory system (Bates, 1989; Dockery and Pope, 1993). A growing body of evidence indicates that particulate pollution increases daily deaths and hospital admissions throughout the world (Pope et al., 1995; Zanobetti et al., 2001). In human risk assessment, the variations in exposure routes (e.g. dermal absorption, inhalation or ingestion) and variation in the sensitivity of different individuals to substances may be considered.

Particulate matters, especially fine particles contain microscopic solids or liquid droplets that are so small that they can get deep into the lungs and cause serious health problems. Various studies conducted in the recent years have linked the particle pollution exposure with many problems like premature death of people suffering from heart or lung diseases, non-fatal heart attacks, irregular heartbeats, asthama, decreased lung function and increased irritation in the respiratory system which includes breathing and coughing as well.

Gaseous co-pollutants, seasonal patterns or weather did not confound the association between particulate pollution and cardiopulmonary mortality (Schwartz, 1994; Samet et al., 2000). Similarly, it was not modified significantly by race, sex and socioeconomic status (Zanobetti and Schwartz, 2000c). Thus, the association between particulate air pollution exposures and cardio-pulmonary mortality appeared causal. The World Health Organization (WHO) has estimated that urban air pollution is responsible for approximately 800,000 deaths and 4.6 million lost life-years each year around the globe (WHO, 2002). It is well recognized that particulate matter PM with an aerodynamic diameter of less than 10 µm (PM_{10}) and less than 2.5 µm ($PM_{2.5}$) are the primary mediators of toxicity in the lungs, while fine ($PM_{2.5}$) and ultrafine particles generally mediate toxicity on the heart and blood vessels (Pope et al., 2002; Brook et al., 2004). People, exposed to toxic air pollutants at sufficient concentrations and durations, may have an increased risk of cancer or experiencing other serious health effects. Therefore, it is important to evaluate $PM_{2.5}$ exposure effect. These considerations have prompted the present study with the objective to determine the $PM_{2.5}$ exposure, through three different exposure pathways and their possible risk assessment in the urban environment of Delhi.

Air Quality Monitoring in Delhi

The daily averaged concentrations of PM2.5 have been obtained from two different air quality monitoring stations namely IGI airport and ITO operated under National Air.

Quality Monitoring Programme (NAMP) (Figure 1). Daily averaged concentration data for $PM_{2.5}$ has been collected from Central Pollution Control Board (CPCB) for the year 2012-2013. National Ambient Air Quality Standard (NAAQS) of daily average concentration for $PM_{2.5}$ is 60 µg/m^3. The concentrations of $PM_{2.5}$ are exceeding NAAQS in 89.25% and 84.2% days during the study period at IGI airport and ITO respectively. The mean and Standard Deviation (SD) of $PM_{2.5}$ is found to be (220.72 ±165.53) µg/m^3 and (133.84 ± 70.61) µg/m^3 at IGI airport and ITO respectively. Similarly the concentrations of $PM_{2.5}$ are exceeding NAAQS in 76.7% and 65.79% days during the rainy days of study period at IGI airport and ITO respectively. Rainy days during the study period have been considered as the deposition of air pollutants affects the concentration of particulate matters. Data corresponding to meteorological parameters has been obtained from two Worlds Meteorological Organization (WMO) stations namely VIDP and VIDD situated in the study domain.

Figure 1.Two different monitoring sites in the study area

Methodology

Human health risk, posed by contaminated air, depends on the potential extent of exposure, as well as the toxic properties of the pollutants. Exposure Assessment is the evaluation of the likely intake of substances. It involves the prediction of concentrations or doses of substances to which the concerned population may be exposed. By considering the different exposure pathways, rate of movement and degradation of a substance can assess exposure. A simple exposure model that we considered throughout the study is:

Exposure = (Concentration × Intake)/ Bodyweight

Where exposure is measured in µg/kg/day, concentration in µg/kg and intake in kg/day and bodyweight in kg.

$PM_{2.5}$ has been considered for hazard identification during the study period 2012-2013. Peoples are exposed to street dust through three main pathways: ingestion of dust particles, inhalation of dust particles and dermal

contact with dust particles. The overall non-cancer risk experienced by human being can be computed for $PM_{2.5}$ by summing the individual risk calculated for each exposure pathways. Although the exposure duration has been set for 5 years (Kushwaha et al., 2012), chronic reference doses are appropriate to evaluate non-carcinogenic risk. Exposure is expressed in terms of a daily dose and is calculated separately for $PM_{2.5}$ and for each exposure pathway.

Dose-Response Assessment

The daily concentration of air pollutants associated with daily morbidity and mortality has been reported in the studies (Levy et al. 1999, Schwartz et al 2001). The US EPA has developed Cancer Slope Factors (CSFs) for carcinogenic effects and an inhalation Reference Concentration (RfDs) for non-carcinogenic effects.

$RfD_{inhalation\ \&\ Ingestion}$ = RfC × 20 m^3 per day / 70 kg = 1.714 x 10^{-2} $mgkg^{-1}day^{-1}$. (1)

Office of Research and Standards (ORS) 1994 recommends using US EPA default exposure factors of 20 m^3 per day respiration rate for 70 kg body weight. (MADEP, 2008 Guidance for Disposal site risks characterization-technical update). RfC is the reference concentration (taken as 60 $\mu g/m^3$).

Average Daily Dose (ADD): The mean amount of an agent to which a person is exposed on a daily basis, often averaged over a long period of time. ADD, which is used for many non-cancer effects can be calculated by averaging the intake dose over body weight and an averaging time as follows:

ADD = (Intake Dose)/(Body Weight * Averaging Time)

Lifetime Average Daily Dose (LADD): Dose rate averaged over a lifetime. The LADD is used for compounds with carcinogenic or chronic effects. The LADD is usually expressed in terms of mg/kg-day or other mass/mass-time units. Often used in carcinogen risk assessments that employ linear low-dose extrapolation methods.

Hazardous Quotient: It is an exposure ratio based on a risk to a human as a receptor being exposed to contaminant via a single pathway, which is expressed as in equation (2).

The hazardous quotient (HQ) = ADD/*RfD* (2)

Where ADD is the average daily dose contacted through different exposure pathway like inhalation, ingestion and dermal and RfD is reference dose.

The RfD is useful as a reference point, which is to measure the potential effects of the species at other doses. Usually, doses less than the RfD is not possibly associated with the adverse health risks and are therefore less likely to be of regulatory concern. As the frequency and magnitude of the exposures exceeding the RfD increase, the possibility of adverse health effects in a human being. However it should not be categorically concluded that all doses below the RfD are acceptable or will be risk free and that all doses in excess of the RfD are unacceptable, which result in adverse health effects.

The inhalation and oral (Ingestion) RfDs are used with average daily doses (ADDs) of contaminants adsorbed to particles to calculate non-cancer health risk.

If HQ < 1, adverse non-cancer health risk is unlikely

> 1, adverse health effect might be possible

> 10, High chronic risk

Following are the three different pathways for non-carcinogenic risk:

Dose contacted through inhalation of street dust is defined in equation (3)

$ADD_{Inhalation}$(mg/kg/day)= (C*InhR*ED)/(BW*AT) (3)

Equation (4) demonstrates the dose absorbed through dermal contact with street dust particles

ADD_{dermal}(mg/kg/day)= (C*SA*SL*ABS*EF*ED)/(BW*AT) (4)

Dose contacted through ingestion of street dust particles has been estimated using equation (5)

$ADD_{Ingestion}$(mg/kg/day)= (C*IngR*EF*ED)/(BW*AT) (5)

Where, C is the concentration of the contaminant in the air (mg/m^3), InhR, inhalation intake rate is taken as 19.92 m^3/day for adult male (Kushwaha et al. 2012); IngR, the ingestion rate, is taken as 200 $mgday^{-1}$ (US Environmental Protection Agency, 2001). EF, the exposure frequency, considered as 350-day yr^{-1}. The average Body Weight (BW) of Indian people is 57 kg for adults (Kushwaha *et al.* 2012). The Exposure Duration (ED) is the length of time that contaminants contact lasts and is calculated by working days (350 days/year) in the service life (5 years) (Kushwaha *et al.* 2012). The Average Time (AT) is 1825 days. The exposed skin area (SA), in this study is taken as 2800 cm^2 (US EPA, 2001), The skin adherence factor (SL) is defined as SL = 0.2 $mgcm^{-2}day^{-1}$ (US EPA, 2001). The dermal absorption factor (ABS) (unitless) is taken as 0.001 (Chang et al., 2009) for $PM_{2.5}$. The Reference Dose (RfD) is the daily exposure below which the adverse non-cancerous health effects are unlikely, if HQ is < 1. The adverse health effects might be possible if HQ > 1. If HQ is > 10, then it suggests a high chronic risk. Although exposure duration has been set at 5 years, chronic reference doses are appropriate to evaluate non-carcinogenic risk. The doses thus calculated for $PM_{2.5}$ through inhalation are subsequently divided by the corresponding reference dose to yield a Hazard Quotient (HQ) (or non-cancer risk).

Lifetime Average Daily Dose (LADD): It is a dose rate, averaged over a lifetime and is used for compounds with

carcinogenic or chronic effects, which is usually expressed in terms of mg/kg-day or other mass/mass-time units. Often used in carcinogen risk assessments that employ linear low-dose extrapolation methods.

For carcinogens, the Lifetime Average Daily Dose (LADD) in the assessment of cancer risk has been calculated as a weighted average for each exposure route as shown in equation (5).

$$LADD\ (mgkg^{-1}day^{-1}) = ADD_{Dermal} + ADD_{Inhalation} + ADD_{Ingestion} \quad (6)$$

Result and Discussion

The doses, thus calculated for each element and exposure pathways are subsequently divided by the corresponding reference dose to yield a Hazard Quotient (HQ) (or non-cancer risk) due to $PM_{2.5}$.

Average Daily Dose (ADD) corresponding to minimum, average and maximum concentration of $PM_{2.5}$ for different exposure pathways at IGI and ITO has been shown in Table 1. Similarly, Average Daily Dose (ADD) corresponding to minimum, average and maximum concentration of $PM_{2.5}$ for different exposure pathways during rainy days at IGI and ITO has been shown in Table 2.

The temporal and spatial variability of the PM 2.5 is influenced by meteorological parameters such as rainfall, temperature, relative humidity, and air flow patterns (Bardouki et al., 2003). Aerosol particles are removed from the air by a combination of wet and dry deposition processes. Wet deposition (precipitation scavenging) occurs in rain events; It is a process, which is rather independent on the particle size, although larger and smaller particles can be removed sequentially during a single rain event.

The Average Daily Dose (ADD) for inhalation corresponding to minimum, average and maximum concentration is quite high in comparison to ingestion and dermal average daily dose. ADD corresponding to average concentration at IGI is 0.068187 $mgkg^{-1}day^{-1}$ higher than 0.041348 $mgkg^{-1}day^{-1}$ at ITO. ADD during rainy days is found to be less in comparison to entire study period (Table 2). This can be attributed to the wet scavenging occurrence in rainy days. Despite wet deposition and wet scavenging daily average concentration of PM2.5 exceeds NAAQS during 76.7% and 65.79% rainy days. HI corresponding to average and maximum concentration of PM2.5 during rainy days is higher than one (Table 3 & 4) at both monitoring site IGI airport and ITO in the study domain.

Table 1.Estimated average daily dose for different pathways (non-carcinogenic risk)

Parameters	Minimum		Average		Maximum	
	IGI	ITO	IGI	ITO	IGI	ITO
ADD (Avg. Daily Dose) InhR (mg $kg^{-1}day^{-1}$)	$2.434x10^{-3}$	$3.358x10^{-3}$	$6.8187x10^{-2}$	$4.1348x10^{-2}$	$2.99103x10^{-1}$	$1.1697x10^{-1}$
ADD (Avg. Daily Dose) Ingestion (mg $kg^{-1}day^{-1}$)	$6.4956x10^{-7}$	$8.9604x10^{-7}$	$1.8194x10^{-5}$	$1.1033x10^{-5}$	$7.9808x10^{-5}$	$3.1212x10^{-5}$
ADD (Avg. Daily Dose) Dermal (mg $kg^{-1}day^{-1}$)	$1.8187x10^{-9}$	$2.5089x10^{-9}$	$5.0943x10^{-8}$	$3.089x10^{-8}$	$2.2346x10^{-7}$	$8.739x10^{-8}$

Table 2.Estimated average aaily aose for aifferent Pathways (non-carcinogenic risk) during rainy days

Parameters	Minimum		Average		Maximum	
	IGI	ITO	IGI	ITO	IGI	ITO
ADD (Avg. Daily Dose) InhR (mg $kg^{-1}day^{-1}$)	$2.4622x10^{-3}$	$5.323x10^{-3}$	$5.3161x10^{-2}$	$2.8672x10^{-2}$	$1.8807x10^{-1}$	$7.52817x10^{-2}$
ADD (Avg. Daily Dose) Ingestion (mg $kg^{-1}day^{-1}$)	$7.3862x10^{-7}$	$7.3862x10^{-7}$	$15.9475x10^{-6}$	$8.6012x10^{-6}$	$56.4197x10^{-6}$	$22.5831x10^{-6}$
ADD (Avg. Daily Dose) Dermal (mg $kg^{-1}day^{-1}$)	$2.0681x10^{-9}$	$4.4708x10^{-9}$	$4.4651x10^{-8}$	$2.4082x10^{-8}$	$1.5797x10^{-7}$	$6.3229x10^{-8}$

Table 3.Hazard quotient and risk for $PM_{2.5}$ and exposure pathway during rainy days

IGI Airport			
Parameters	**Minimum ADD**	**Average ADD**	**Maximum ADD**
$HQ_{Inhalation}$	0.1436	3.1016 > 1	10.973 > 1
$HQ_{Ingestion}$	0.000043	0.00093	0.00329
HQ_{Dermal}	0.00000012	0.0000026	0.0000092
Hazard Index (HI)	0.1436	3.981 > 1	10.455 > 1

Table 4.Hazard quotient and risk for $PM_{2.5}$ and exposure pathway during rainy days

ITO			
Parameters	**Minimum ADD**	**Average ADD**	**Maximum ADD**
$HQ_{Inhalation}$	0.3105	1.6728 > 1	4.3922 > 1
$HQ_{Ingestion}$	0.00004308	0.0005017	0.001317
HQ_{Dermal}	0.0000002608	0.0000014	0.00000368
Hazard Index (HI)	0.31054334	1.6733 > 1	4.39352 > 1

HI corresponding to average concentration is higher than one, during rainy days, showing the persistence of higher level of $PM_{2.5}$ concentration at both monitoring sites in the study area. RfD for Inhalation and Ingestion (Oral) is taken as calculated by (eq.1). Since, the Reference dose of dermal exposure for $PM_{2.5}$ is not available, this can be replaced by the reference dose of Ingestion (Guide to Regulation of Toxic chemicals, Dec 1990). Further HQ and HI through three different exposure pathways at IGI and ITO have been shown in Table 5 & 6 respectively. It is clear from table 5 and 6 that HQ corresponding to inhalation is very high in comparison to ingestion and dermal at IGI and ITO respectively. Higher HQ and consequently larger HI at IGI in comparison to ITO (Table 2 and 3) can be attributed to the higher $PM_{2.5}$ concentration at IGI airport.

Table 5.Hazard quotient and risk for $PM_{2.5}$ and exposure pathway

IGI Airport			
Parameters	**Minimum ADD**	**Average ADD**	**Maximum ADD**
$HQ_{Inhalation}$	0.142	3.98 > 1	17.45 > 1
$HQ_{Ingestion}$	0.000038	0.001061	0.004655
HQ_{Dermal}	0.00000011	0.00000297	0.00001303
Hazard Index (HI)	0.142	3.981 > 1	17.455 > 1

Table 6.Hazard quotient and risk for $PM_{2.5}$ and exposure pathway

ITO			
Parameters	Minimum ADD	Average ADD	Maximum ADD
$HQ_{Inhalation}$	0.196	2.412 > 1	6.824 > 1
$HQ_{Ingestion}$	0.000052	0.00064	0.00182
HQ_{Dermal}	0.00000015	0.0000018	0.0000051
Hazard Index (HI)	0.196	2.413 > 1	6.825 > 1

The IGI airport is more susceptible to pollution than the city side because it has large open spaces. The open spaces have lesser green patches due to which the dust particles get settled in the air, as compared to the other areas of Delhi. Pollutants from the highly polluted Gurgaon region and Dwarka also make their entry into the air surrounding the airport, raising its pollution level. The high pollution levels at the airport are a combination of the very high traffic intensity in and around the airport. Due to the busiest airport in India, it has an influx of Air Traffic Movements (ATMs) as well as the motorized vehicles around it. Whereas, ITO the major traffic intersection site, in the study area experiencing consistently higher level of $PM_{2.5}$ apart from the peak traffic hours throughout the day.

The major limitations of the present study are also discussed:

- The daily averaged concentrations of air pollutants monitored at IGI airport and ITO are used in the present study. These two locations have been chosen on the basis of availability of data more than 80% during the year.
- In the present study, reference dose for dermal exposure of $PM_{2.5}$ is replaced by the reference dose of ingestion for $PM_{2.5}$, However, the same dermal exposure would be estimated if sampling of the dermal doses have been available in the study area.
- Unavailability of concerned health data for the study area.
- The model could be evaluated with other studies based on similar type of data. However, the study cannot be validated, since the observed data is not available.

Conclusion

It is found that $PM_{2.5}$ exceeds NAAQS of (60 µg/m^3) for 89.25% and 84.2% of the total days of the study year at IGI airport and ITO monitoring stations respectively. The Mean (SD) of $PM_{2.5}$ is estimated as (220.72 ± 165.53) µg/m^3 and (133.84 ± 70.61) µg/m^3 at IGI airport and ITO respectively, which are observed to be much more than the national ambient air quality standards. Average Daily Dose of Inhalation of $PM_{2.5}$ corresponding to mean and maximum concentrations is found to be higher at IGI than that of ITO. But the minimum average daily dose is found to be higher at ITO. Hazard Quotient associated with the inhalation of $PM_{2.5}$ is observed to be maxima among all the three different exposure pathways (Inhalation, Ingestion and Dermal) considered. However the Hazard Index (HI) associated with $PM_{2.5}$ through three different exposure pathways corresponding to mean daily average concentration and maximum concentration is found to be higher at IGI airport in comparison to ITO. The hazard index corresponding to ADD at IGI and ITO is found to be 3.981 and 2.413 respectively. As HI is more than 1, it indicates that human health is severely affected by the presence of higher concentration of $PM_{2.5}$ at both the places. According to the above analysis of HQ, the human health risk associated with $PM_{2.5}$ is resulted more at IGI airport than ITO (Traffic intersection site).

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