



## Human health risks in national capital territory of Delhi due to air pollution

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### ABSTRACT

This study evaluates the human health risks in Indian National Capital Territory of Delhi (NCT Delhi) in terms of mortality and morbidity due to air pollution. The spreadsheet model, Risk of Mortality/Morbidity due to Air Pollution (Ri-MAP) was used to evaluate the direct health impacts of various criteria air pollutants present in various districts of NCT Delhi during the period 1991 to 2010. By adopting the World Health Organization (WHO) guideline concentrations for the air pollutants SO<sub>2</sub>, NO<sub>2</sub> and total suspended particles (TSP), concentration–response relationships and a population attributable–risk proportion concept were employed. About 11 394, 3 912, 1 697 and 16 253 excess number of cases of total mortality, cardiovascular mortality, respiratory mortality and hospital admission of COPD respectively were observed for entire NCT Delhi in year 2000. However, within a one decade, in year 2010 these figures became 18 229, 6 374, 2 701 and 26 525. District–wise analysis shows that North West district is having the highest number of mortality and morbidity cases continuously after 2002, moreover least excess number of cases was observed for New Delhi district.

**Keywords:** Air Pollution, mortality, morbidity, human health, respiratory diseases



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### 1. Introduction

In the Indian National Capital Territory of Delhi (NCT Delhi), air pollution (e.g. PM, NO<sub>x</sub>, CO etc.) levels have exceeded most other cities in developing countries since the early 1990s (Gurjar et al., 2004; Nagpure, 2011). Scientific studies have indicated that the air pollution is mostly caused by combustion sources and causes human health impacts ranging from respiratory illnesses to death in urban areas (Mage et al., 1996; Gurjar et al., 2004; Madronich, 2006; Butler et al., 2008; Kumar et al., 2008; Gurjar et al., 2010; Kumar et al., 2011; Nagpure et al., 2011; Nagpure and Gurjar, 2012; Nagpure et al., 2013). Estimating the health impacts could help initiating international and national efforts to improve the air quality of NCT Delhi by implementing new policies and norms (Gurjar et al., 2008; Nagpure et al., 2010; Shukla et al., 2013; Babae et al., 2014). To this end, Gurjar et al. (2008) calculated trace gas and particle emissions and ambient air quality in the Multi-Pollutant Index (MPI) to reveal air pollution impacts in the world's 18 largest megacities. Likewise, Gurjar et al. (2010) used average values of ambient concentration of various air pollutants (e.g. SO<sub>2</sub>, PM, NO<sub>x</sub>) to estimate health risks in terms of mortality and morbidity in NCT Delhi. However, limitations of the Gurjar et al. (2010, 2012) studies were that they (1) did not address direct health impacts occurring annually due to particular pollutants, and (2) used average values of air pollutants for the entire city rather than at a district level, which produced general city–level results. Indeed, district level or area–wise ambient air quality monitoring and health risk modeling helps to identify least and most vulnerable areas with respect to air pollution within the megacity

or state. Current study is the extension and expansion of our previous study (Gurjar et al., 2010). This study focuses on the quantification of various pollutants (SO<sub>2</sub>, NO<sub>2</sub> and TSP) to assess human health impacts (mortality/morbidity) in various districts of NCT Delhi covering the areas of North–West, South, West, North–East, South–West, East, North, Central and New Delhi districts during the period 1991 to 2010 using the Risk of Mortality/Morbidity due to Air Pollution (Ri-MAP) model. A limitation to this study is the lack of analysis on (1) Synergistic effects of two or more pollutants, which is important because some pollutants can be more harmful when paired with others, and (2) other environmental and nutritional factors, which could cause greater sensitivity to poor air quality.

### 2. Material and Methods

#### 2.1. Background of relative risk

In epidemiology, the relative risk (RR) is the probability of developing an illness caused by the exposure to pollutants (WHO, 2003; Rothman et al., 2008) The World Health Organization (WHO) has specified RR values and corresponding baseline incidences for different air pollutants as well as types of diseases associated with those values (Table 1). WHO (2003) relevant input data files in the air quality health impact assessment software AirQ2.2 (URL: <https://euro.sharefile.com/download.aspx?id=s7fd1f822339468c8>) was adopted for this study. The data is based on various previous studies (e.g., Poloniecki et al., 1997; Burnett et al., 1997; Spix et al., 1998; Sunyer, 1997; Touloumi, 1997; WHO, 2000).

**Table 1.** WHO default values of relative risk (per 10 µg/m<sup>3</sup> increase of daily averages for SO<sub>2</sub>, TSP and NO<sub>2</sub>) corresponding to mortality

Pollutant	Mortality/Morbidity	Relative Risk (RR)	Baseline Incidence Per 100 000 (I) <sup>b</sup>
SO <sub>2</sub>	Total Mortality	1.004 (1.003–1.0048) <sup>c</sup>	1 013
	Cardiovascular Mortality	1.008 (1.002–1.012)	497
	Respiratory Mortality	1.010 (1.006–1.014)	66
	Hospital Admission COPD <sup>a</sup>	1.0044 (1–1.011)	1 014
TSP	Total Mortality	1.003 (1.002–1.007)	1 013
	Cardiovascular Mortality	1.002 (1–1.006)	497
	Respiratory Mortality	1.008 (1.004–1.018)	66
	Hospital Admissions COPD <sup>a</sup>	1.0044 (1–1.0094)	1 014
NO <sub>2</sub>	Total Mortality		
	Cardiovascular Mortality	1.002 (1–1.004)	497
	Respiratory Mortality		

Note: The total, cardiovascular and respiratory mortality is the annual number of deaths in a given age group per the population in that age group (usually expressed per 100 000) due to exposure of air pollution. However Hospital Admission COPD is the annual number of morbidity in a given age group per the population in that age group (usually expressed per 100 000) due to exposure of air pollution.

<sup>a</sup> COPD: Chronic Obstructive Pulmonary Disease

<sup>b</sup> Baseline Incidence per 100 000 is based on threshold limit given in WHO guideline

<sup>c</sup> Lower and upper limits (range) of the 95% confidence interval of RR values

## 2.2. Concentration response equations

Estimation of health impacts from air pollutants is based on the population attributable–risk proportion (AP) concept (Douwes et al., 2002; Rothman et al., 2008). The assessment of mortalities in Ri–MAP model is based on long term exposure (1 year) to the air pollutants (PM, NO<sub>x</sub>, SO<sub>2</sub>) whereas the morbidity is based on short term exposure (24 hours) (WHO, 1999; WHO, 2003). Assuming that there is a causal relationship between exposure and health outcomes with no major confounding effects, the AP can be attributed to pollutant exposure in a given population for a certain time period. This can be calculated using the following equation (WHO, 1999):

$$AP = \frac{\sum\{[RR(c) - 1] \times p(c)\}}{\sum[RR(c) \times p(c)]} \quad (1)$$

where,  $RR(c)$  is the changed relative risk for the health outcome in category  $c$  of exposure and  $p(c)$  is the proportion of the population in category  $c$  of exposure which could vary according to the degree of exposure in a different area. For example, industrial and residential areas have contrasting degrees of exposure, so health impacts from pollutants would vary. However, we must assume the same exposure in all districts throughout the city due to the lack of data availability.

$$RR(c) = \frac{(C - T)}{10} \times (RR - 1) + 1 \quad (2)$$

where,  $C$  is the ambient air concentration of a pollutant,  $T$  is the threshold level of the pollutant as recommended by the WHO, and  $RR$  is the relative risk for the selected health outcome, which can be derived from the exposure–response function gathered from local epidemiological studies. We used the WHO default values because local studies for all districts were not available (Table 1).

Exposure of various populations to pollutants was measured using monitoring data from stations within NCT Delhi (see the Supporting Material, SM, Figure S1). We used the arithmetic mean of selected concentrations for each time unit (daily or yearly). The average value was then used as indicator of the whole population's exposure (i.e. one population – one value for a specified time period). For this purpose, we used daily concentrations data from 9 monitoring stations and taken the yearly average value of them.

Knowing (or often assuming) a certain baseline frequency (at threshold concentration value given by WHO guideline) of selected health outcomes (i.e.,  $I$ ) and the rate (or number of cases per unit population) attributed to the exposure in population (i.e.,  $IE$ ) can be calculated as (WHO, 1999):

$$IE = I \times AP \quad (3)$$

For a population of given size  $N$ , the  $IE$  can be converted to the estimated number of cases attributed to exposure (i.e.,  $NE$ ) using the following equation:

$$NE = IE \times N \quad (4)$$

Consequently, the frequency of the outcome in the population that is free from exposure (i.e.,  $INE$ ) can be estimated using the following equation:

$$INE = I - IE = I \times (1 - AP) \quad (5)$$

After deriving the  $RR$  at a certain level of pollution and the estimated incidence in non–exposed population, the excess incidence [i.e.,  $\Delta I(c)$ ] and excess number of cases [i.e.,  $\Delta N(c)$ ], respectively, at a certain category of exposure ( $c$ ) can be calculated using the following equations:

$$\Delta I(c) = (RR(c) - 1) \times p(c) \times INE \quad (6)$$

$$\Delta N(c) = \Delta I(c) \times N \quad (7)$$

All aforementioned formulas are based on the assumption that the  $RR$  estimate is adjusted for any possible confounding variables. When the limits of the confidence interval for the  $RR$  estimate are used in the first equation, we obtain the corresponding AP range and the respective range for the number of cases in the population that can be attributed to pollutant exposure. The last equation is used to calculate the excess number of morbidity cases, which denotes the number of mortalities in the exposed population. In practice, however, the uncertainty of the impact (and the range of the estimated effect) is greater due to errors in exposure assessment and non–statistical uncertainty of the exposure–response function (WHO, 1999; WHO, 2003).

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