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ARTICLE

Short and long-term exposure to ambient air pollution and impact on health in India: a systematic review

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ABSTRACT

Health effects attributable to short-term and long-term ambient air pollution (AAP) exposure in Indian population are less understood. This study evaluates the effect of short time and long-term exposure to AAP on respiratory morbidity, mortality and premature mortality for the exposed population. A total of 59 studies are reviewed to examine the effects of short-term exposure (n = 23); long-term exposure (n = 18) and premature mortality (n = 18). Short-term exposures to ambient pollutants have strong associations between COPD, respiratory illnesses and higher rates of hospital admission or visit. The long-term effects of AAP, associated with deficit lung function, asthma, heart attack, cardiovascular mortality and premature mortality have received much attention. Particulate matter ($PM_{2.5}$ and PM_{10}) is primarily responsible for respiratory health problems. Out of 18 literature reviewed on premature mortality, most (12 of 18) studies have statistically significant associations between AAP exposure and increased premature mortality risk.

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KEYWORDS
Ambient air pollution (AAP); particulate matter (PM); respiratory health; mortality

Introduction

A well-established body of literature, documents and reports highlights how both short- and long-term exposure to ambient [outdoor] air pollution (AAP) (mainly consisting of nitrogen oxides [NOx], sulfur dioxide [SO_2], particulate matter [PM], ozone [O_3] and carbon monoxide [CO]) is associated with adverse health outcomes (Liu et al. 2013; Sehgal et al. 2015; Kesavachandran et al. 2015b; Cohen et al. 2017; Haque and Singh 2017). The health effects are measured by short-term exposure to pollutants by hours, days or weeks and long-term exposure of months or years (Beverland et al. 2012). The effects of short-term exposure on human health include exacerbation of pre-existing respiratory disease especially asthma, chronic obstructive pulmonary disease [COPD], chronic respiratory sputum, cough, wheezing problem, breathing problem and pre-existing cardiovascular disease (including ischemia, arrhythmias and cardiac failure). This leads to an increase in hospitalization and emergency department visits (Kumar et al. 2004; Gupta 2008; Patankar and Trivedi 2011; Maji et al. 2015, 2018). The longer and more intense the exposure greater is the impact on health, ranging from minor eye irritations, cough, wheezing, allergic rhinitis, respiratory symptoms to decreased lung and heart function, tuberculosis, cardiovascular diseases and even premature death (Kumar and Foster 2007; Guttikunda and Jawahar 2012; Guttikunda and Goel 2013; Ghosh and Mukherji 2014; Tobollik et al. 2015; Gawande et al. 2016).

In India, millions of people breathe high concentrations of dreaded pollutants (Thambavani and Vathana 2012). This has led to greater chances of associated health effects on the population manifested in the form of sub-clinical effects, impaired pulmonary functions, use of medication,

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reduced physical performance, frequent medical consultations and hospital admissions with complicated morbidity and even death in the exposed population. Respiratory and cardiovascular diseases are the most documented health effects associated with poor air quality (Maji et al. 2018). Among major health risk factors in India, AAP ranked 5th in mortality and 7th in overall health burden (PHFI and CEH, 2017). It is estimated that AAP leads to approximately 670,000 deaths annually (Dholakia et al. 2014). According to the World Health Organization (WHO), 14 cities of India feature in the top 20 world's most polluted cities in terms of particulate matter <2.5 μm in diameter ($PM_{2.5}$). The cities of Kanpur, Faridabad, Varanasi, Gaya, Patna, Delhi, Lucknow, Agra, Muzaffarpur and Srinagar are listed in the top 10 polluted cities of the world (WHO 2019). Rapid industrialization and increasing economic activity across the country have resulted in worsening of AAP in these Indian cities.

In addition, nearly 76% of rural households are dependent on solid biomass as cooking fuels and thus experience household air pollution (HOAP) and the burden of AAP (Balakrishnan et al. 2013). Such exposure greatly exceeds the WHO Air Quality Guideline (AQG) levels (Balakrishnan et al. 2013; Gordon et al. 2018). The rapid growth in the industrial, power and transportation sectors, combined with rapid urbanization (both planned and unplanned) have contributed to the rapid increase in AAP levels in India. Together, the substantial growth in the number of automobiles and coal-based power production trash burning, unregulated use of personal diesel generator sets and brick kilns are expected to significantly contribute to the worsening of air quality in the next decade in India (Mannucci and Franchini 2017; Gordon et al. 2018).

In this systematic review, we collected literature with different study designs, these studies were carried out in different cities of India from January 1st, 1990 to 31 January 2019. For, example: epidemiological time series and case-crossover studies were conducted in Delhi (Cropper et al. 1997; Chhabra et al. 2001; Pande et al. 2002; Rajarathnam et al. 2011; Maji et al. 2018); Kanpur (Gupta 2008); Ludhiana (Kumar et al. 2010), Chennai (Public Health Foundation of India and centre for environmental health 2017); Mumbai, Shimla, Ahmedabad, Bangalore and Hyderabad (Dholakia et al. 2014). Apart from the above-mentioned studies, few had also investigated the effects of air pollution on health outcomes. Therefore, we conducted a systematic review of AAP exposure for both shorter and longer time periods in order to understand and quantify the burden of diseases and mortality for the exposed population. Our hypothesis was that an increase in particulate matter (PM_{10} and $PM_{2.5}$), ozone (O_3), carbon monoxide (CO), nitrogen oxide (NO_2 and NO_X) and sulfur dioxide (SO_2), is associated with an adverse health impact (morbidity/mortality) in India.

Methods: search strategy and selection criteria

For this systematic review and meta-analysis, we searched Taylor and Francios, Science Direct, J-STOR, Research Gate, Scopus, Book SC, Google Scholar and PubMed. We limited our search to studies published between 1 January 1990 and 31 January 2019, and to studies in English language only. Also, we considered peer-reviewed original articles. The search strategies included all possible combinations of keywords from the following two groups: (1) ambient air pollution, long-term exposure, short-term exposure, ambient air pollutants, particulate matter, fine particulate matter, sulfur dioxide, ozone, carbon dioxide, carbon monoxide, suspended particulate matter and nitrogen oxide [PM, $PM_{2.5}$, PM_{10}, SO_2, O_3, CO_2, CO, SPM, NO_2] (2) respiratory morbidity, hospital visit, hospital admission, respiratory mortality, cardiovascular mortality and premature mortality. We also manually searched the references of most of the studies for additional publications. Further publications were also identified by examining the review articles. We applied no study design restrictions to the search. To eliminate irrelevant articles in search results were screened by title and by abstract.

The inclusion and exclusion criteria for this review

We include only those articles which examined the adverse effect of ambient air pollutants (PM2 · 5 [PM with a diameter of <2 · 5 μm] and PM_{10} [PM with a diameter of 2 · 5–10 μm] NO_2, CO_2, SPM, SO_2, O_3, CO, etc.) on human health. Also, studies on India or Indian states or cities were considered for inclusion. To maintain the focus on AAP, we excluded household or indoor sources of air pollution-based study. We excluded abstracts of conference presentations because they did not contain sufficient information. Also, studies explaining only ambient air pollution without the inclusion of health impact were excluded. In addition, those studies which examine the health risk by joint exposure (household air pollution and ambient air pollution) of air pollution were also excluded. Those articles fulfilling all inclusion and exclusion criteria were included in the list of literature for further review (Figure 1).

Data extraction and statistical analysis

Data were extracted by the author and recorded in an access database. The following information was extracted from each included study: name of the first author, publication year, follow-up period, study design, pollutant type, location of the study, study population, demographics, number of cases (exposed and non-exposed or control) health outcome, adjusted variable, statistical model and estimates odds ratio [OR], regression coefficient (b), risk ratio (RR), and their corresponding 95% confidence interval [CI] or standard error.

We performed a meta-analysis to estimate the overall effect of ambient air pollutants (PM2.5 and PM_{10} NO_2, CO_2, SPM, SO_2, O_3, CO, etc.) on the composite outcome of any respiratory

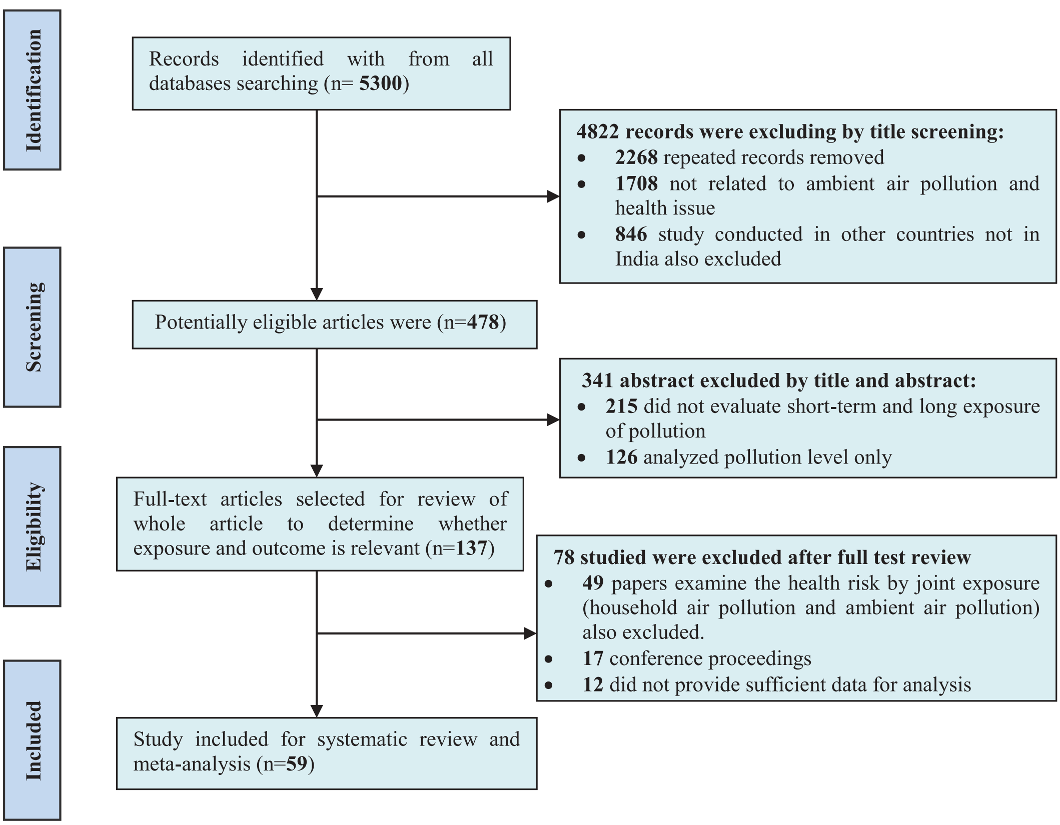

Figure 1. Flow chart of the study selection process.

Table 1. WHO specified default value of Relative Risk (RR) per 10 ug/m$_3$ increase of daily average for PM_{10}, SO_2& NO_2 with 95% Confidence Intervals (CI).

Pollutants	Mortality/Morbidity	Relative risk (RR)[b] (95% CI) per 10 ug/m^3	Baseline incidence per 100,000[c]
PM_{10}	Total Mortality [x]	1.0074 (1.0062–1.0086)	1013
	Cardiovascular Mortality [y]	1.008 (1.005–1.018)	497
	Respiratory Morbidity [z]	1.012 (1.008–1.037)	66
	Hospital admission respiratory diseases	1.008 (1.0048–1.0112)	1260
	Hospital admission Cardiovascular diseases	1.009 (1.006–1.013)	436
SO_2	Total Mortality [x]	1.004 (1.003–1.0048)	1013
	Cardiovascular Mortality [y]	1.008 (1.002–1.012)	497
	Respiratory Morbidity [z]	1.01 (1.006–1.014)	66
	COPD[a] Morbidity (hospital admission)	1.0044 (1–1.011)	101.4
NO_2	Cardiovascular Mortality [y]	1.002 (1–1.004)	497
	COPD Morbidity (hospital admission)	1.0038 (1.004–1.0094)	101.4

Source: Nagpure et al. (2014); Maji et al. (2016)

morbidity (cough, sneezing, COPD, sputum, wheezing problem, breathing problem, cold, short time illness, bronchitis, asthma and other possible respiratory diseases) and to account for both within- and between-study heterogeneity. The statistical software STATA (Version 14, STATA Corporation) was used to generate forest plots and pooled Odds Ratio (ORs) for groups of respiratory morbidity. Meta-analysis in STATA (Version 14) was performed using the "metan" command.

Result

Our results sections are divided into four categories: the first section deals with respiratory morbidity due to exposure of AAP, the second section reveals long-term exposure of AAP mainly on lung function and mortality, the third section is empirical evidence of AAP on premature mortality, and the fourth section we applied Meta-analysis to see the relationship between AAP and respiratory morbidity.

In epidemiological terms, the relative risk (RR) is the probability of developing diseases caused by the exposure to pollutants (Nagpure et al. 2014). The World Health Organization (WHO) has specified RR values and corresponding baseline incidences for different air pollutants as well as morbidities/mortalities associated with those values (Table 1).

a COPD: Chronic obstructive Pulmonary diseases; **b** Lower and upper limits (range) of RR value; **c** Baseline incidence 100,000 is based of threshold limit is given in WHO guideline; **x** International classification of diseases (ICD) code number: ICD-9-CM<800; **y** ICD-9-CM 390–459; **z** ICD-9 460–519.

Short-term effect of AAP

The health effects of short-term exposure to air pollution are very important aspects of environmental epidemiology (Szyszkowicz 2018). The vast majority of epidemiological studies reporting health effects of AAP in India used data from urban centers and reported on the prevalence of respiratory morbidity. Through a systematic review, we identified eight cross-sectional studies; seven case-control studies, five time-series studies, two cohorts and one case study with respect to short-term effect (See Table 2). These studies are investigating the association between ambient exposure and respiratory morbidity, hospital admission/hospital visit and emergency room visit for asthma or other respiratory causes (Chhabra et al. 2001; Pande et al. 2002; Kumar et al. 2004; Ingle et al. 2005; Jayanthi and Krishnamoorthy 2006; Ghose 2009; Patankar and Trivedi 2011; Centra l Pollution Control Board 2012; Liu et al. 2013; Kesavachandran et al. 2015b; Prasad and Sanyal 2016; Cohen et al. 2017; Haque Singh, 2017; Maji et al. 2018). Most of these existing studies

Table 2. Summary of selected epidemiologic studies between the study period 1990–2019.

Study design	Short-term	Long-term	Premature mortality	Total
Time series	5	5	6	16
Cross-sectional	8	9	-	17
Case-control	7	2	-	9
Cohort study	2	1	6	9
Case study	1	1	1	3
Modelling/bottom-up modelling study	-	-	4	4
Intervention study	-	-	1	1
Total	**23**	**18**	**18**	**59**

on AAP and impact on health are based on the physical linkage approach, where a dose–response function is estimated in order to observe the relationship between human health and air pollution. These studies largely concentrated in metropolitan cities like Delhi, Mumbai, Chennai and Kolkata and found that elevated levels of AAP are the main causes of prevalence of diminished lung function, acute and chronic respiratory symptoms (cough, wheeze and asthma) in children and adults (Gordon et al. 2018; Sehgal et al. 2015; Gawande et al. 2016; Public Health and Air Pollution in Asia [PAPA], 2011; Ghose 2009; Chhabra et al. 2001; Pande et al. 2002).

Interestingly, the short-term exposure resulting from acute increases in $PM_{2.5}$ and PM_{10} is not limited to the critically ill or dying. In fact, much of the morbidity and mortality occurred among active individuals are with one or more risk factors. Nagpure et al. (2014) observed about 11,394 (total mortality), 3912 (cardiovascular mortality), 1697 (respiratory mortality) and 16,253 (hospital admission of COPD) deaths, respectively, for entire NCT Delhi in the year 2000. However, within one decade, in the year 2010, these figures became 18,229, 6374, 2701 and 26,525, respectively. At national level, Cohen et al. (2017) also explained that exposure to $PM_{2.5}$ caused 29,609.6 thousand (25,923.3–33 562.7) disability-adjusted life-years (DALYs) in India during 2015. From the systematic review, it is clear that if air pollutants ($PM_{2.5}$, PM_{10}, SO_2, NO_2 and CO_2) cross their daily prescribed standards level (each 10-µg/m3 or 50-µg/m3) for the long run, it increases the risk of the burden of disease.

Ambient air pollution (AAP) and respiratory health

There is a significant effect of AAP on respiratory illnesses such as allergies, asthma, COPD and lung cancer (Chhabra et al. 2001; Patankar and Trivedi 2011; Gawande et al. 2016; Prasad and Sanyal 2016). Although associations between AAP and respiratory disease are complex, a recent cross-sectional study by Patankar and Trivedi (2011) estimated that there was a net increase of cough by 0.35% if PM10 is 50 $\mu g/m^3$ increased. Similarly, breathlessness increase by 1.40% if NO_2 increased by 20 $\mu g/m^3$. Similarly, COPD increases by 1.15% if NO_2 increased 20 $\mu g/m^3$. Another cross-sectional study by Kumar et al. (2004) has shown that the prevalence of chronic respiratory symptoms (cough, breathlessness, or wheezing) was 27.9% and 20.3% in the industrial town and reference towns (non-industrial town), respectively ($p < 0.05$). Epidemiological studies indicate that increased risk for cardiovascular diseases, lung cancer and respiratory diseases (bronchitis and respiratory tract infections) in urban residents compared to a rural population (Vimercati 2011; Central Pollution Control Board 2012a; Ghose 2009). A study by Central Pollution Control Board (2012a), found a similar risk among school children in Delhi. The findings were compared with rural school children in West Bengal. It was found that Delhi had 1.7 times higher prevalence of respiratory symptoms (in the past 3 months) compared with rural controls of West Bengal, i.e. ($P < 0.001$). The odds ratio of upper respiratory symptoms in Delhi was 1.59 (95% CI: 1.32–1.91) times more and for lower respiratory symptoms (a dry cough, wheeze, breathlessness, chest discomfort) it was 1.67 (95% CI: 1.32–1.93) times more in Delhi compared to West Bengal. In a similar study conducted in Kolkata city by Ghose (2009), it is found that air pollution levels in urban Kolkata were several times higher than rural West Bengal. After controlling for tobacco chewing and passive smoking (the presence of smokers in the family) as possible confounders, authors found that due to AAP respiratory symptoms increased 1.98 (CI: 1.29–3.66), lung function was reduced 2.23 (CI: 1.67–4.15), and siderophage numbers substantially increased 4.43 (CI: 2.54–7.68) respectively. Aggarwal et al. (2006) reported significant associations between respiratory ailments (COPD, asthma and emphysema) with pollutants (SO_2, NO, SPM and RSPM) and meteorological factor exposure. The authors reported that a positive statistical correlation exists between COPD cases and SPM ($r = 0.474$; $p < 0.01$), RSPM ($r = 0.353$; $p < 0.05$). Also, from correlation analysis, it can be concluded that while the two pollutants SPM and RSPM have a direct bearing on the number of COPD cases. However, if a meteorological variable like temperature is minimum, it has a significant inverse effect on the same. A study conducted in the state of Texas found that major predictor of hospitalization due to asthma was season; it was highest in winter period when ozone levels are usually lowest (Nnoli et al. 2018). Sagar et al. (2007) examined the air pollution-related morbidity of highly polluted area (HPA) and low polluted area (LPA) of Delhi. About 23% of asthma patient was found in the highly polluted area (HPA) as compared to 9% patient in a low populated area (LPA). Allergy and dermatitis were also more in HPA (15.9%) than in LPA (4.7%) population [Table 3].

Ambient air pollution (AAP) and hospital visit/admission

Short-term exposure to air pollutants were significantly associated with hospital admission and emergency-room visits due to asthma, chronic obstructive pulmonary disease (COAD), acute coronary event (ACE) and COPD, pediatric respiratory diseases (PRD), upper respiratory infection (URI) and lower respiratory infection (LRI) (Pande et al. 2002; Jayaraman et al. 2008; Liu et al. 2013). Pande et al. (2002) found that due to high level of outdoor pollution, the emergency room visits for asthma increased by 21.30%; chronic obstructive airways disease increased by 24.90% and acute coronary events (ACE) increased by 24.30%. Liu et al. (2013), explained that there is a strong correlation between visits to a hospital due to respiratory disease and AAP. According to him, a relative number of patients who visited the hospital per number of inhabitants in each pollution zone (high to low zone) is much higher in the highly polluted regions than in the less polluted regions. The result showed that risk of hospital admission was changed according to the pollution zone. Polluted and less polluted zones have 1.64 odds ratio (CI: 1.43–1.89); very highly polluted and less polluted zones have 3.25 odds ratio (CI: 2.80–3.78) and very highly polluted to highly polluted zones have 0.98 odds ratio (CI: 0.87–1.09) of hospital admission. Also, from other studies, it was proved that higher-pollution zone has a higher chance of respiratory morbidity 2.30 odds ratio (CI: 1.74–3.05) as compared to lower pollution zone 2.21 odds ratio (CI: 1.80–2.71) (Chhabra et al. 2001). Maji et al. (2017) found the significant associations between gaseous air pollutants (NO_2, O_3 and NO) and hospital admissions for COPD cases. Findings revealed that hospitalization due to COPD cases was increased over the years. In 2002, the attributable number of estimated cases was 138 (95% CI: 76–197) followed by 186 (95% CI: 106–261) in 2006, 153 (95% CI: 90–213) in 2010 and 167 (95% CI: 99–232) in 2014. Another study by Jayaraman et al. (2008) determined the association between the air pollutants and daily variations in respiratory morbidity and hospital admission in Delhi. The authors found that admissions for asthma, pneumonia and influenza were significantly associated with NO_2, O_3 and RSPM. Relative risk (RR) for admissions for the respiratory disease for three pollutants ranged from 1.033 (1.019–2.770) for O_3, 1.004 (0.992–2.696) for NO_2, 1.006 (1.004–2.728) for RSPM, respectively. Apart from particulate matter (PM) other criterion air pollutants like O_3 and CO also affect respiratory health. From recent time series study we found that for hospital admission due to respiratory disease, largest effect estimate was observed for O_3 (above 65 $\mu g/m^3$) to be associated with 3.41% (CI: 0.02–6.83) increase in hospital admission rate (Maji et al. 2018).

Table 3. Short-term exposure of AAP and its impact on respiratory morbidity and mortality.

Author	Follow-up period	Pollutant	Outcome measure	Exposed population, characteristics & pollution exposure	Result & effect (95% CI)
Chhabra et al. 2001	1990–2000	TSPs, SO_2, NO_2	Bronchial, Asthma, chronic bronchitis, & COPD	Low SES = M: 716, F: 524; Middle SES = M: 815, F: 699; High SES = M: 813, F: 574.	Higher-pollution zone: **2.30 (1.74, 3.05)**; Lower pollution zone: **2.21 (1.80, 2.71)**
Kumar et al. 2004	1999–2001	TSP, PM, SO_X & NO_x,	Sputum, Cough, Wheezing problem & Breathing problem	Study Town: M: 1105, F: 722; Reference Town: M: 908, F: 868. (M = Male, F = Female)	Chronic respiratory symptoms risk: **1.5 (1.2–1.8)** Spirometric ventilatory defect risk: **2.4 (2.0–2.9)**
Ingle et al. 2005	2003–2004	SPM, SO_x, NO_x, & CO	Cough, Shortness of breath, RTI	60 Male in exposed group and 60 Male in control group	Frequent coughing **2.96 (2.4–3.6)**
Padhi and Padhy 2008	2006–2007	NO_2, SO_2, TSPM	Asthma and respiratory disorders, Lung Function	750 young male adults, Investigated peak expiratory flow rate, IUATLD based questionnaire	Those living 5.0 km away from a main road to those subjects living within 0.5 km and 1.0 km had OR of **1.00 (0.85 to 1.50), 3.57 (3.00 to 3.95)**, and **3.00 (2.85 to 3.50)**, respectively for doctor-diagnosed asthma.
Agarwal et al. 2006	2000–2003	SO_2, NO, SPM and RSPM	COPD, asthma and emphysema	Health exposure data collect from hospital record. Pollution data from CPCB,& IMD which provided with the meteorological data of the study area.	Positive correlation of COPD with SPM **(r = 0.474; p < 0.01)** Positive correlation of COPD with RSPM **(r = 0.353; p < 0.05)**. Positive correlation was observed between COPD and Asthma **(r = 0.514; p < 0.01)**.
Sagar et al. 2007	2006	SPM RSPM TSPM, NO2 and SO2	Respiratory Morbidity	A total of 640 subjects participated in the study. [High pollution area (N = 320)] and [Low pollution area (N = 320)]	Respiratory infection **86 (26.9) <0.001**, Breathlessness **112 (35) <0.001**, Irritation of throat **136 (42.5) <0.001**. Cough: **220 (68.8) 0.4**, Sneezing **168 (52.5) 0.008**, Common cold **158 (49.4) 0.0003**, Burning eyes: **124 (38.8) <0.001**
Patankar and Trivedi 2011	2003–2004	PM_{10} and NO_2	Cough, cold, breathlessness, wheezing and illnesses such as allergic rhinitis and COPD	1542 respondents (11+ years of age) Government	Cough = **0.35** net effect if PM_{10}, 50 μg/m^3 is increase. Breathlessness = **1.40** net effect if NO_2 20 μg/m^3 increase. COPD **= 0.70** net effect if PM10, 50 μg/m^3 is increase.
Ghose 2009	2008–2009	SPM, RPM, SO_2, NO_x, & PM_{10}.	Nose block, Sneezing, Cough & Hyperacidity	450 Male in exposed group and 410 Male in control group	Increased respiratory symptom: **1.98 (1.29–3.66)**, Reduced lung function: **2.23 (1.67–4.15)**, Higher siderophage numbers: **4.43 (2.54–7.68)**
Central Pollution Control Board, 2012a	2002–2005	SPM, PM_{10}, SO_2, & NO_2	Lung function, attacks of shortness of breath, wheezing, dry or wet cough and chest tightness.	A total number of 16,164 children aged between 4–17 years participated in this study.	Dry cough: **1.48 (1.24–1.67);** Wet cough: **1.33 (1.12–1.56);** Wheeze: **1.23 (1.04–1.45);** Breathlessness on exertion: **1.37 (1.15–1.63)**. Medically diagnosed asthma: **1.08 (0.87–1.42)**; Eye irritation: **2.45 (1.56–3.74)**

(Continued)

Table 3. (Continued).

Author	Follow-up period	Pollutant	Outcome measure	Exposed population, characteristics & pollution exposure	Result & effect (95% CI)
Gawande et al. 2016	2013	SO_2, NO_x, CO_2 SPM, H_2S	Respiratory diseases and lung function among children	2000 school children in the age group 6–7 years and 13–14 years were selected	Dry Cough: **29.5%** in Study and **21.3%** in Control, p = 0.000 Significant by Chi Square test Night Cough: **22.3%** in Study and **13.6%** in Control, p = 0.000 Significant by Chi Square test Sneezing:**17.8%** in Study and **10.2%** in Control, p = 0.000 Significant by Chi Square test
Prasad and Sanyal 2016	2008–2011	PM_{10}, $PM_{2.5}$, SPM, SO_2, NOx and CO	Air-borne diseases (Asthma, Bronchitis, Pneumonia, TB, Lung Cancer, Congenial Heart), Suffocation,	Household survey (14 sample stations in residential, commercial and industrial areas)	SPM impact on Asthma R **= .821 (p = .045)** RSPM impact on Asthma R = **.856 (significant = .029)**SPM impact on Bronchitis R = **.839(significant = .037)** RSPM impact on Bronchitis R = **.896 (significant = .016)** RSPM Congested Heart R **= .859(significant = .029)**
Nagpure et al. 2014	1991 2010	SO_2, NO_2 and TSP	Total mortality, cardiovascular mortality, respiratory mortality and hospital admission of COPD respectively.	Population data obtained from Census of India (2001, 2011). The ambient atmospheric concentrations of criteria pollutants monitored and estimated by the CPCB, Delhi. Health risks have been calculated for various districts of the NCT Delhi using ambient air pollution concentration data of particular districts. Each district is used for calculating district–wise health risk estimates.	About **11,394, 3912, 1697** 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.
Cohen et al. 2017	1990–2015	$PM_{2.5}$ and Ozone	IHD, cerebro vascular disease, lung cancer, COPD and lower respiratory infections (LRI), and the burden attributable to ozone for COPD	Global population-weighted mean concentrations of particle mass with aerodynamic diameter less than 2 · 5µm ($PM_{2.5}$) and ozone at an approximate 11 km × 11 km resolution with satellite-based estimates	Deaths, in thousands (95% UI) **1090 · 4(936 · 6–1254 · 8)** DALYs, in thousands (95% UI): 29 **609 · 6 (25 923 · 3–33 562 · 7)**

SES socioeconomic status; **PM_{10}** Particulate matter ≤10 µm in aerodynamic diameter; **$PM_{2.5}$** Particulate matter ≤2.5 µm in aerodynamic diameter; **NO_2** Nitrogen dioxide; **O_3** Ozone; **SO_2** Sulfur dioxide; **TSP** Total suspended particles; **CO** Carbon monoxide; **RSPM** Respirable Suspended Particulate Matter; **SPM** suspended particulate matter; **RPM** respirable particulate matter **TSPM** Total Suspended Particulate Matter; **H_2S** Hydrogen Sulfide; **TB** Tuberculosis; **IHD** Ischaemic heart disease; **RTI** Respiratory Tract Infection **CPCB** Central pollution control board; **IMD** India Meteorological Department; **DALY** Disability-Adjusted Life Year; **IUATLD** International Union against Tuberculosis and Lung Disease.

A notable study by Gupta (2008) estimated the monetary benefits of individuals from health damages avoided due to reductions in air pollution. He found that the annual welfare gains to a working individual from reduced air pollution are Rs 130.39 due to the reduction in workdays lost and due to the reduced medical expenditures is Rs 34.43 per person. This, constitute a total gain of Rs 212.82 million per annum of the study population. Indeed, hospital admissions and emergency room visits due to respiratory morbidity have been recognized as a sensitive marker than mortality for assessment of the air pollution effects on human health (Gupta 2008; Liu et al. 2013) [Table 4].

Long-term effect of AAP

Long-term exposure to air pollution has been associated with chronic bronchitis, markers of atherosclerosis, respiratory impairment, lung cancer and mortality (Cropper et al. 1997; Kumar et al. 2010; Public Health and Air Pollution in Asia [PAPA], 2011; Balakrishnan et al. 2013; Ghosh & Mukherjee 2014; Tobollik et al. 2015; Gawande et al. 2016; & Maji et al. 2016). There is a significant number of new studies on long-term air pollution exposure, covering a wider range of the geographic regions of India. These recent studies support the positive associations between ambient pollutants (PM_{10}, NO_2 & SO_2) and high risk of mortality. Overall, the available studies provide evidence that long-term exposure to AAP in India is associated with asthma cases, reduced lung function, lung cancer, non-trauma death, cardiovascular deaths, respiratory deaths and premature death (Cropper et al. 1997; Pandey et al. 2005; Sehgal et al. 2015 & Gawande et al. 2016). The pooled effect estimate that the excess risk per 10 μg/m^3 increase in PM_{10} exposure was 1.004 (1.002 to 1.007) for all-cause mortality in Tamil Nadu, and for Delhi, it was 1.0015 (1.0007 to 1.0023) [PAPA, 2011].

Another study suggests that long-term exposure to outdoor particulate air pollutant (PM_{10}) significantly increased the risk of pneumonia in children, i.e. 1.26 OR (CI: 1.00–1.57) for 6 μg/m^3 increase in the concentration (Jiang et al. 2018). Also, ambient NO_2 was a consistent risk factor for doctor-diagnosed asthma (OR 1.19 per 10 μg/m^3), doctor diagnosed rhinitis (OR 1.28 per 10 μg/m^3), lifetime eczema (OR 1.16 per 10 μg/m^3) and current wheeze (OR 1.13 per 10 μg/m^3) among pre-school children (Norback et al. 2019). A prospective cohort study indicated that the risk of childhood allergic rhinitis was associated with traffic-related air pollutant, NO_2 during entire pregnancy with OR 1.38 (CI: 1.03–1.84) in single-pollutant model and OR 1.69 (CI: 1.03–2.77) in multi-pollutant model (Deng et al. 2016). In addition, case fatality also depends on age, pre-existing cardiovascular disease, obesity, low socioeconomic status smoking and other individual factors. Individual susceptibility to the health effects of air pollution may differ, due to either biological differences or behavioral differences affecting exposure (Stockfelt 2017).

Ambient air pollution (AAP) and its effect on lung function

Lung function is an important measure of respiratory health, and it may be compromised by repeated exposure to contaminated air. AAP frequently causes exacerbation and deterioration of both asthma and COPD and may be a potential cause for lung cancer. Of the most common ambient air pollutant, particulate matter (PM) is associated with an increased risk of exacerbations and respiratory symptoms in individuals with existing lung disease, and to a lesser extent, in those without known respiratory issues (Adam et al. 2014). However, coarse soil dust particles (size range of 3–8 μm) with low density, could be deposited deeper in the lungs (pulmonary region) than the traffic particles (size range of 0.08–0.7 μm) which are fine with high density (Deng et al. 2019). Over the past two decades, researchers worldwide have investigated the long-term effects of AAP on lung function with most finding adverse effects (Siddique et al. 2010a). Pervious study found that each 10-μg/m^3 increase in long-term ambient $PM_{2.5}$ concentrations is associated with a 12% increased risk of acute lower respiratory infections (ALRI) incidence. Also, the decrease of

Table 4. Short-term exposure of AAP on hospital visit/admissions.

Author	Follow-up period	Pollutant	Outcome measure	Exposed population, characteristics &pollution exposure	Result & effect (95% CI)
Pande et al. 2002	1997–1998	TSP, CO, SO_2 & NOx	Hospital visit, Emergency room visit for asthma, COAD & ACE	Total cardio-respiratory events: 6478 cases (In 1997), 9334 cases (in 1998) COAD: 1240 cases (in 1997), 1944 cases (in 1998) Asthma: 2138 cases (in 1997) 3151 cases (in 1998) ACE: 3100 cases (1997), 4239 cases (in 1998).	**Asthma**, observed **(7.23 + 7.82)**; expected **(5.96 + 4.55)** and increase **21.30 percent. COAD**, observed (**4.37 + 4.97);** expected **(3.50 + 2.48)** and increase **24.90 percent ACE**, observed **(10.09 + 2 7.09)**; expected **(8:11 + 3.07)** and increase **24.30 percent**
Gupta 2008	2004	SO_2, NOx, & SPM	Household health, Number of days of illness, Hospitalization, doctor's consultation.	Selection of Households (HHs): The Low class category: 385 HHs. The lower middle class category: 116 HHs. High Class category: 104 HHs	Workdays lost due to Asthma = **3.5390 (2.20)** 1 μg/m^3 fall in RSPM results in a marginal gain of **0.00007** for a representative person in a week.
Jayaraman and Nidhi, 2008	2004–2005	O_3, NO_2 and RSPM	Daily hospital visits over the study period.	Daily count of patients with respiratory diseases visiting the respiratory unit of a hospital. Air pollution data obtained from CPCB and the meteorological data of Delhi obtained from the India Meteorological Department.	10 μg m^{-3} rise in pollutant level led to statistically significant relative risks (RR): **1.033 (1.019–2.770)** for O_3, **1.004 (0.992–2.696)** for NO_2, **1.006(1.004–2.728)** RSPM
Liu et al. 2013	2006	SO_2, PM and NOx	8,340 patients who visited the hospital at least once in the study period	Hospitalization, Hospital visit (specialist pulmonary hospital with symptoms of respiratory disease), cardiopulmonary diseases	Polluted and less polluted cluster: **1.64 (1.43, 1.89)**; Very highly polluted and polluted cluster: **1.98 (1.76, 2.22)**; Very highly polluted and highly polluted: **0.98 (0.87, 1.09)**
Maji et al. 2016	2004–2013	PM_{10}, SO_2, and NO_2	Hospital admissions and Respiratory disease and hospital admissions cardiovascular Disease (HACD).	City based study (Mumbai, Pune, Nagpur, Thane, Nashik, Aurangabad, Solapur, Navi Mumbai, Kolhapur, Chandrapur)	**ENC of Hospital admission** Mumbai: **20,955 (17,689–24,028)** Pune: **3,502 (2,939–4,040)** Nagpur: **812 (681–939)** Thane: **413 (347–478)** Nashik: **(754–1,047)** Aurangabad: **497 (419–574)** Solapur: **837 (710–960)** Navi-Mumbai: **952 (803–1,093)** Kolhapur: **251 (211–289)** Chandrapur: **175 (147–203)**
Maji et al. 2018	2008–2012	PM_{10}, NO_2, SO_2, CO, O_3	Hospital admission as well as OPD visit due to respiratory and cardiovascular illnesses	Respiratory OPD patient: 14,785; Cardiovascular OPD patient: 13,183; Respiratory IPD patient: 394	The risk estimates of hospital admission with respiratory problem was estimated to be **0.47 (CI: 0.03–0.91) per 10 μg/m^3 increase in 75th percentile of daily average PM_{10}** concentration for a month.

COAD Chronic obstructive pulmonary disease; **ACE** acute coronary event **ENC** Excess number of cases **PM_{10}** Particulate matter ≤10 μm in aerodynamic diameter; **$PM_{2.5}$** Particulate matter ≤2.5 μm in aerodynamic diameter; **NO_2** Nitrogen dioxide; **O_3** Ozone; **SO_2**Sulfur dioxide; **TSP** Total suspended particles; **CO** Carbon monoxide; **RSPM** Respirable Suspended Particulate Matter; **SPM** suspended particulate matter.

10-μg/m^3 of PM would be safe to Years of Life Lost (YLL) (Mehta et al. 2013). A research conducted by Lin (Lin et al. 2018) showed that exposure to $PM_{2.5}$ was associated with a total of 454.6 years of life lost (YLL) (95% UI 449.0–460.1) per 100,000 people in the year 2009. Similarly, Kumar and Foster (2007) found that one standard deviation increase in current $PM_{2.5}$ results in a 0.28 standard deviation reduction in lung function. Also, one standard deviation increase in current $PM_{2.5}$ results in a 0.628 point decline in lung function. Siddique et al. (2010a) explained that increasing levels of PM_{10} are associated with increased lung function deficits of obstructive and restrictive type. Studies reveal that urban children are the most vulnerable group in this regard. The level of PM_{10} in ambient air is associated with obstructive 1.45 OR (CI: 1.16–1.82), restrictive 1.35 OR (CI: 1.07–1.58) and combined type of lung function deficits 1.74 OR (CI: 1.37–2.71) in children. Another study by Arora et al. (2018) commented that smokers and participants with a history of respiratory disease had 4.619 OR (CI: 1.075–19.851) and 3.479 OR (CI: 1.121–10.798) higher chances at ($P < 0.05$) of abnormal deficits lung function (Table 5).

Ambient air pollution (AAP) and mortality

A recent study published in Lancet, (2018) found that in 2017, 1.24 million (95% UI 1.09–1.39) deaths in India were attributable to air pollution. The number of deaths attributable to ambient particulate matter (APM) pollution in India in 2017 was 0.67 million (95% UI 0.55–0.79). Also, the point estimate for the number of deaths attributable to APM pollution among males in India in 2017 (0.39 million [95% UI 0.32–0.46]) was 38.3% higher than for females (0.28 million [0.22–0.34]) (Lelieveld et al. 2018). Also, Cohen et al. (2017) estimated that in 2015, long-term exposure to ambient $PM_{2.5}$ caused 1090.4 (95% UI 936.6–1254.8) thousand death and 29,609.6 (95% UI 25,923.3–33 562.7) lost years of healthy life in India. We also found that long-term exposure to $PM_{2.5}$ was more associated with mortality from cardiovascular disease (particularly ischemic heart disease) and lung cancer than from non-malignant respiratory diseases. Each 100 μg/m^3 increase in total suspended particles (TSP) is associated with 2.3% change in total non-trauma death; 4.3% change in cardiovascular mortality and 3.1% change in respiratory infection (Cropper et al. 1997). A similar result is found by Balakrishnan et al. (2013) estimated 0.44% (95% CI 0.17–0.71) increase in mortality per 10 μg/m^3 increase in daily average concentrations of PM_{10}. On the other hand, Tobollik et al. (2015) found that a decrease of 10% in PM concentrations would save 15,904 (95% CI: 11,090–19,806) life years. A city-based study by Maji et al. (2017) observed that cardiovascular death due to pollutants (PM_{10}, SO_2 and NO_2) in Agra city was 908 (95% CI: 412–1372) in 2002 followed by 1193 (95% CI: 559–1771) in 2006, 1004 (95% CI: 474–1496) in 2010 and 1095 (95% CI: 517–1632) in 2014. Overall, the available studies provide evidence that long-term exposure to AAP in India is associated with asthma cases, reduced lung function, lung cancer, non-trauma death, cardiovascular deaths, respiratory deaths and premature death (Cropper et al. 1997; Pandey et al. 2005; Sehgal et al. 2015; Gawande et al. 2016) [Table 6]. At the national level, an absolute number of deaths from COPD attributable to ozone increased from 43,500 thousand in 1990 to 1.80 million in 2015 [Table 7].

Ambient air pollution (AAP) and premature mortality

Over the past decades, numerous epidemiological studies and meta-analysis, estimating increased premature mortality due to short- and long-term exposure to PM (Guttikunda and Jawahar 2012; Silva et al. 2013; Ghude et al. 2016). In India, about 0.62 million premature excess number of death cases occurred due to AAP and became the 5th leading cause of death after high blood pressure, indoor air pollution, tobacco smoking and poor nutrition in 2012 (Maji 2016). Chowdhury and Dey (2016) estimate of 486,100 (73,200–1,254,800) annual premature deaths in India but it is lower than some of the recent estimates for India as part of global study (Pope et al.

Table 5. Long-term effects of AAP on lung function.

Author	Follow-up period	Pollutant	Outcome measure	Exposed population, characteristics & pollution exposure	Result & effect (95% CI)
Kumar and Foster 2007	2003–2004	$PM_{2.5}$	To collect respiratory health and socio economic data, 1576 household's survey. Also CPCB data used.	Respiratory health and Lung function	One standard deviation increase in $PM_{2.5}$ exposure results in a **0.79-point** decline in lung function. A one standard deviation increase in $PM_{2.5}$ results in a **0.628** point decline in lung function.
Patel et al. 2008	2007	SOx, NOx, SPM	Total 2,573 female residents selected for the medical examination. Out of that 415 participants selected for pulmonary function test. Also, GPCB data used.	Respiratory symptoms and pulmonary function test.	Pulmonary Function Test Abnormalities (restrictive type) **2.03 (0.70–5.93)** of living in Vapi, in town, or in villages proximal to the Gujarat Industrial Development Corporation.
Siddique et al. 2010a	2002–2005	SPM, RSPM, NO_2 & SO_2	Exposed group consisted of 5,671 children (3,708 boys and 1,963 girls). The control group 2,245 children (1,438 boys and 807 girls).	Reduced lung function. Restrictive, obstructive and combined type of lung functions deficits.	Level of PM (76–100 μg/m^3) **RR 1.34 (1.06–1.68)** Level of PM (101–125 μg/m^3) **RR 1.62 (1.40–1.87)** Level of PM (>125 3.75 μg/m^3) **RR (3.50–4.60 μg/m^3)**
Sehgal et al. 2015	2014	$PM_{2.5}$, CO, NO_2 & SO_2.	153 workers respondents aged below 40 years with no smoking history.	Lung function, respiratory impairment, Asthma and Heart attack	Difference between means of lung function duration of work 3–5 years **0.74 (0.25–1.33)** duration of work >5 years **0.94 (0.48–1.40)**
Kesavachandran et al. 2015b	2014	PM_{10} and $PM_{2.5}$	433 men & 421 women living in the National Capital Region [Noida & Gurgaon] participated in the study. Ambient air quality monitoring was conducted 10 randomly chosen locations.	Lung function (FEV1) test.	The prevalence of airway obstruction cases in women **(OR, 1.96, 95% CI; 1.42–2.71)** was higher **($p < 0.001$)** among female subjects compared to their male counterparts.
Khafaie et al. 2017	2015–2016	PM_{10}	400 exposed group (diabetic patient) and 465 non-exposed group (healthy people)	Chronic symptoms and the pulmonary function tests (PFT)	1 SD increase in PM_{10} was associated with a greater risk of having dyspnea by **1.50 (1.12–2.01)**.The size of effect for 1 SD μg/m^3 increase in PM10 concentration was **3.71 (0.48–4.99)** decrease in FVC.
Arora et al. 2018	2015	NO_2, PM_{10} and $PM_{2.5}$	Out of 550 adult women aged 18–59 years in 528 houses, eventually,500 females gave consent for participation in the study.	Altered lung function	Smokers and participants with a history of respiratory disease had **4.619 (1.075–19.851)** and **3.479 (1.121–10.798)** at $P < $ **0.05** higher chance of abnormal forced expiratory volume in 1 (FEV1).

PM_{10} Particulate matter ≤10 μm in aerodynamic diameter; **$PM_{2.5}$**Particulate matter ≤2.5 μm in aerodynamic diameter; **NO_2**Nitrogen dioxide; **O_3**Ozone; **SO_2**Sulfur dioxide; **TSP** Total suspended particles; **CO** Carbon monoxide; **RSPM** Respirable Suspended Particulate Matter; **SPM** suspended particulate matter.

Table 6. Long-term effects of AAP on mortality.

Author	Follow-up period	Pollutant	Outcome measure	Exposed population, characteristics &pollution exposure	Result & effect (95% CI)
Cropper et al. 1997	1991–1994	TSP, SO_2, & NO_x	NDMC Non-Trauma Deaths in different board Municipal Corporation of Delhi Urban: 34,455 New Delhi Municipal Committee: 1,999Delhi Cantonment Board: 49	Non-trauma death, Cardiovascular Deaths & Respiratory Deaths	100 Microgram increase in TSP the Total Non-trauma Deaths was **2.3** increased and Cardiovascular Deaths was **4.3** increased (significance at 95% CI).
Kumar et al. 2010	2002–2004	RSPM, NO_x and SO_2	Overall in the 3- year period, 28,007 deaths were registered, with an average of 25.4 deaths per day.	Daily death due to air pollutants	Effect of air quality on mortality **1.007 (1.002–1.013)**
PAPA, 2011	2002–2004	PM_{10}, NO_2 & SO_2	**Chennai**: Daily all-cause mortality ranged from 60 to 229 deaths in a year, **Delhi**: Total daily all-cause mortality varied from 126 to 368 with an average of 222 deaths per day.	Daily all-cause mortality	**Tamil Nadu**: **1.004 (1.002 to 1.007)** **Delhi**: **1.0015 (1.0007 to 1.0023)**
Balakrishnan et al. 2013	2002–2004	PM_{10}	Data on daily total all-cause mortality were collected from the Chennai City Corporation office. (Daily mortality: N = 1096)	Daily all-cause mortality	**0.44 (0.17–0.71)** increase in mortality per 10 μg/m^3 increase in daily average concentrations of PM_{10} Relative risk (RR) **1.0044 (1.002–1.007).**
Dholakia et al. 2014	2005–2012	PM_{10}	Daily all-cause mortality data were collected from the birth and death registers of the municipal corporations of Ahmedabad, Bangalore, Hyderabad, Mumbai and Shimla.	Daily all-cause mortality	Ahmedabad **0.16 (0.31 to 0.62)** Bangalore **0.22 (0.04 to 0.49)** Hyderabad **0.85 (0.06 to 1.63)** Mumbai 0.20 **(0.10 to 0.30)** Shimla **1.36 (0.38 to 3.1)**
Tobollik et al. 2015	2008–2011	PM	Natural Deaths per 100,000 People Male: 635, Female: 401 Cardiovascular Deaths per 100,000 People Male: 194, Female: 139	Cardiovascular Deaths, premature mortality	Life years were lost due to PM2.5: Male: **58,868 (40,003–75,094)** Women: **37,490 (25,476–47,823)**
Maji et al. 2017	2002–2014	PM_{10}, SO_2 and NO_2	Population data in 2001 and 2011 have been taken from Census of India. Pollution data from CPCB, 2015	Total mortality, cardiovascular mortality and respiratory mortality	Cardiovascular death **908 (95% CI: 412–1372)** in 2002, **1193 (95% CI: 559–1771)** in 2006, **1004 (95% CI: 474–1496)** in 2010 and **1095 (95% CI: 517–1632)** in 2014.

NDMC New Delhi Municipal Corporation**: CPCB**Central Pollution Control Board **PM_{10}** Particulate matter ≤10 μm in aerodynamic diameter; **$PM_{2.5}$**Particulate matter ≤2.5 μm in aerodynamic diameter; **NO_2**Nitrogen dioxide; **O_3**Ozone; **SO_2**Sulfur dioxide; **TSP** Total suspended particles; **CO** Carbon monoxide; **RSPM** Respirable Suspended Particulate Matter; **SPM** suspended particulate matter.

Table 7. Trends of morbidity, mortality and DALYs due to AAP in India during 1990–2015.

	1990		1995		2000		2005		2010		2015	
Morbidity, mortality and DALYs	Total cases	Global share (%)	Total cases	Global share (%)	Total cases	Global share (%)	Total cases	Global share (%)	Total cases	Global share (%)	Total cases	Global share (%)
Numbers of deaths attributable to PM2.5 exposure	7,37,400	21.21	7,95,200	21.76	8,57,300	22.60	8,95,900	22.77	9,57,000	24.26	10,90,400	25.71
Numbers of deaths from COPD attributable to exposure to ozone for all ages and sexes.	43,500	27.50	54,400	29.84	66,800	33.62	73,000	35.27	88,100	40.54	1,07,800	42.44
Age standardized DALY rates (DALYs/100,000 people) due to PM2.5 exposure	4,100	(2500/ 100,000 people)	3,900	(2300/ 100,000 people)	3,700	(2100/ 100,000 people)	3,300	(1900/ 100,000 people)	3,000	(1600/ 100,000 people)	2,900	(1500/ 100,000 people)

Source: The State of Global Air, 2017
d. Meta-analysis of respiratory morbidity

2002; Cohen et al. 2005; Apte et al. 2015) because of the difference in methodology. Forouzanfar et al. (2016) using a raw estimate of $PM_{2.5}$ calculated annual premature death of 587,000 for India. Even at the state level, the scenario of premature death has variation, Chowdhury and Dey (2016) identified that Indian states of Uttar Pradesh, Bihar, West Bengal, Maharashtra and Delhi metropolitan area are most vulnerable and contribute 25%, 15%, 7.6%, 5.4% and 1.7% to total all- India premature death from ambient $PM_{2.5}$ exposure in the previous decade. Guttikunda et al. (2014) explained that due to high concentrations of $PM_{2.5}$, the premature deaths in greater Chennai and greater Vishakhapatnam regions were 4850 and 1250, respectively. For comparison, Guttikunda and Jawahar (2012) estimated deaths of 3600 for Pune, 4950 for Ahmedabad, 1800 for Indore and 1250 for Surat. For cities similar in area and size, the estimated premature mortality was 7350 to 16,200 premature deaths for Delhi in 2010 (Guttikunda and Goel 2013).

Our systematic reviews suggest that $PM_{2.5}$ considerably impact premature mortality and life expectancy in India. In fact, the economic cost of estimated premature mortalities associated with $PM_{2.5}$ and O_3 exposure is about 640 (350–800) billion USD in 2011, which is a factor of 10 higher than total expenditure on health by the public and private expenditure (Ghude et al. 2016) [Table 8].

We observed very high heterogeneity between the selected studies (I^2 = 92.3%); hence the random effect model was used to analyze and obtain the pooled RR for the studies. Table 9 presents the calculated RR and their 95% confidence interval along with the weights assigned to the studies (Inverse variance method for weighting the studies). The pooled RR for the prevalence of respiratory disease is estimated to be 1.75 odds ratio (CI: 1.46–2.10). In other words, individuals exposed to pollution are 75% more likely to suffer from any respiratory disease. The forest plot of the RR of selected studies shows that only one study has a negative relationship and rest shows the positive relationship (Figure 2). Heterogeneity from the pooled estimate is also evident from the figure.

The funnel plot of the selected studies is asymmetric with higher studies showing the positive relationship between AAP and respiratory morbidities (Figure 3). Further, it can be noted that the majority of the studies are significant at 1% of the level of significance (P = <0.01) with only one study having negative relation and only one turned insignificant (Figure 4).

To assess the publication bias and effect of a sample on the result, Egger's meta-regression model is applied. Through the above-mentioned model, it is observed that there is no sample size effect on the estimate. Hence, it can be interpreted that small studies, as well as large studies, would show similar results.

Discussion

Ambient air pollution (AAP) is linked not only with diseases that kill but also with poor health and morbidity among millions of children (UNICEF 2016) as well as adult population. In evaluating the literature, there appears to be a consistent and significant effect of AAP on human health. Short-term exposures to ambient particulate and gaseous pollutants have already shown strong associations between chronic obstructive pulmonary disease (COPD) (Chhabra et al. 2001; Agarwal et al. 2006; Patankar and Trivedi 2011), respiratory illnesses (Sagar et al. 2007; Gupta and Elumalai 2017) and higher rates of hospital admission/visit (Pande et al. 2002). While the long-term effects of air pollution have been associated with deficit lung function (Kumar and Foster 2007; Siddique et al. 2010a; Arora et al. 2018), asthma (Sehgal et al. 2015), heart attack (Sehgal et al. 2015), cardiovascular mortality (Tobollik et al. 2015) and premature mortality (Ghose 2009; Guttikunda and Jawahar 2012; Silva et al. 2013; Guttikunda and Kopakka 2014; Guttikunda and Jawahar 2014; Lelieveld et al. 2015; Ghude et al. 2016; Chowdhury and Dey 2016; Dey 2018). Also, particulate matter ($PM_{2.5}$ and PM_{10}) is primarily responsible for deleterious health problems, including asthma, bronchitis, chronic obstructive pulmonary disease, pneumonia, upper respiratory tract and lower respiratory tract disorders.

Table 8. Estimated premature mortality due to outdoor air pollution in India.

State/region/City	States	Study year	Pollutant	Premature mortality	Projection of premature mortality	Study design	Reference
National level estimation							
All India	National Level	1990	PM_{10}	438,000	-	Time series	IHME 2013
All India	National Level	2010–2011	$PM_{2.5}$	80,000–115,000	-	Time series	Guttikunda and Jawahar 2014
All India	National Level	2010	$PM_{2.5}$ and O_3	644,993	-	Cohort Study	Lelieveld et al. 2015
All India	National Level	2010	PM and Ozone	69,000	-	Time series	OECD, 2014
All India	National Level	2011	$PM_{2.5}$	570,000	-	Modelling Study	Ghude et al. 2016
All India	National Level	2013	$PM_{2.5}$	397 000	-	Cohort Study	Silva et al. 2013
All India	National Level	2016	$PM_{2.5}$	486,100	-	Cohort Study	Chowdhury and Dey 2016
All India	National Level	2031–2040	$PM_{2.5}$		9100–12,300 Per year	Cohort Study	Chowdhury et al. 2018
Northern India	North India	2018	O_3	37,800	-	Cohort Study	Karambelas et al. 2018
State level estimation							
State Level	Uttar Pradesh	2011	$PM_{2.5}$	85,500	-	Bottom-up Modelling Study	Ghude et al. 2016
State Level	West Bengal	2011	$PM_{2.5}$	51,300	-	Bottom-up Modelling Study	Ghude et al. 2016
State Level	Maharashtra	2011	$PM_{2.5}$	57,000	-	Bottom-up Modelling Study	Ghude et al. 2016
State Level	Bihar	2011	$PM_{2.5}$	45,600	-	Bottom-up Modelling Study	Ghude et al. 2016
City level estimation							
Delhi	National Capital	2001	PM_{10}	5000	-	Time series	Nema & Goyal 2010
Kolkata	West Bengal	2001	PM_{10}	4300	-	Time series	Nema & Goyal 2010
Mumbai	Maharashtra	2001	PM_{10}	2000	-	Time series	Nema & Goyal 2010
Chennai	Tamil Nadu	2001	PM_{10}	1300	-	Time series	Nema & Goyal 2010
Ahmedabad	Gujarat	2001	PM_{10}	4300	-	Time series	Nema & Goyal 2010
Kanpur	Uttar Pradesh	2001	PM_{10}	3200	-	Time series	Nema & Goyal 2010
Surat	Gujarat	2001	PM_{10}	1900	-	Time series	Nema & Goyal 2010
Pune	Maharashtra	2001	PM_{10}	1400	-	Time series	Nema & Goyal 2010
Bhopal	Madhya Pradesh	2001	PM_{10}	1800	-	Time series	Nema & Goyal 2010
Mumbai	Maharashtra	2000	PM_{10}	454	-	Case Study	Joseph et al. 2003
Kolkata	West Bengal	2010	SPM	10,000	-	Case-Control Design	Ghose 2009
Pune	Maharashtra	2010	PM_{10}	3600	4300 (In the year of 2020)	Intervention Study	Guttikunda and Jawahar 2012
Chennai	Tamil Nadu	2010	PM_{10}	3950	6000 (In the year of 2020)	Intervention Study	Guttikunda and Jawahar 2012
Indore	Madhya Pradesh	2010	PM_{10}	1800	2500 (In the year of 2020)	Intervention Study	Guttikunda and Jawahar 2012
Ahmedabad	Gujarat	2010	PM_{10}	4950	7850 (In the year of 2020	Intervention Study	Guttikunda and Jawahar 2012
Surat	Gujarat	2010	PM_{10}	1250	2050 (In the year of 2020)	Intervention Study	Guttikunda and Jawahar 2012
Rajkot	Gujarat	2010	PM_{10}	300	670 (In the year of 2020)	Intervention Study	Guttikunda and Jawahar 2012
Delhi	National Capital	2010	$PM_{2.5}$ & PM_{10}	7,350–16,200	-	Bottom-up Modelling Study	Guttikunda and Goel 2013
Hyderabad	Telangana	2010–11	$PM_{2.5}$	3700	-	Modelling Study	Guttikunda and Kopakka 2014
Varanasi	Uttar Pradesh	2011	PM_{10}	5700	-	Time series	Jain &Chowdhury, 2017
Chennai	Tamil Nadu	2012	$PM_{2.5}$	4850	-	Time series	Guttikunda et al. 2015
Vishakhapatnam	Andhra Pradesh	2012	$PM_{2.5}$	1250	-	Time series	Guttikunda et al. 2015
Agra	Uttar Pradesh	2016	$PM_{2.5}$	2421	-	Cohort Study	Dey, 2018

(*Continued*)

Table 8. (Continued).

State/region/City	States	Study year	Pollutant	Premature mortality	Projection of premature mortality	Study design	Reference
Allahabad	Uttar Pradesh	2016	$PM_{2.5}$	1443	-	Cohort Study	Dey, 2018
Gaya	Bihar	2016	$PM_{2.5}$	710	-	Cohort Study	Dey, 2018
Gorakhpur	Uttar Pradesh	2016	$PM_{2.5}$	914	-	Cohort Study	Dey, 2018
Kanpur	Uttar Pradesh	2016	$PM_{2.5}$	4173	-	Cohort Study	Dey, 2018
Lucknow	Uttar Pradesh	2016	$PM_{2.5}$	4127	-	Cohort Study	Dey, 2018
Meerut	Uttar Pradesh	2016	$PM_{2.5}$	2044		Cohort Study	Dey, 2018
Muzaffarpur	Bihar	2016	$PM_{2.5}$	531		Cohort Study	Dey, 2018
Patna	Bihar	2016	$PM_{2.5}$	2841	-	Cohort Study	Dey, 2018
Ranchi	Jharkhand	2016	$PM_{2.5}$	1096	-	Cohort Study	Dey, 2018
Varanasi	Uttar Pradesh	2016	$PM_{2.5}$	1581	-	Cohort Study	Dey, 2018

Table 9. Risk ratio estimates for respiratory morbidity due to ambiant air pollution: meta-analysis of selected studies (n = 16).

Study ID	Authors and Year	RR	95% CI		Weight
1	Ingle et al. 2005	2.118	1.347	3.329	5.01
2	Kumar et al. 2004	1.429	1.192	1.714	6.77
3	Ghose 2009	1.755	1.353	2.278	6.32
4	CPCB (a), 2012	1.679	1.523	1.85	7.11
5	Khafaie et al. 2017	2.399	1.8	3.197	6.15
6	Siddique et al. 2010b	1.847	1.512	2.256	6.68
7	Kesavachandran et al. 2015b	1.016	0.674	1.532	5.3
8	Singh et al. 2009	1.796	1.107	2.916	4.79
9	Gupta et al. 2011	2.72	1.888	3.919	5.62
10	Singh et al. 2018	5.459	3.81	7.822	5.66
11	CPCB (b) et al. 2012	1.685	1.483	1.915	7.01
12	Krishnan et al. 2019	2.184	1.907	2.501	6.98
13	Gawande et al. 2016	1.385	1.189	1.614	6.91
14	Sagar et al. 2007	2.175	1.656	2.857	6.24
15	Patel et al. 2008	0.545	0.439	0.676	6.59
16	Nair et al. 2011	1.502	1.273	1.771	6.85
	D + L pooled RR	**1.751**	**1.459**	**2.10**	**100.00**

(Chaimani et al. 2014) https://ebmh.bmj.com/content/ebmental/17/4/111.full.pdf.

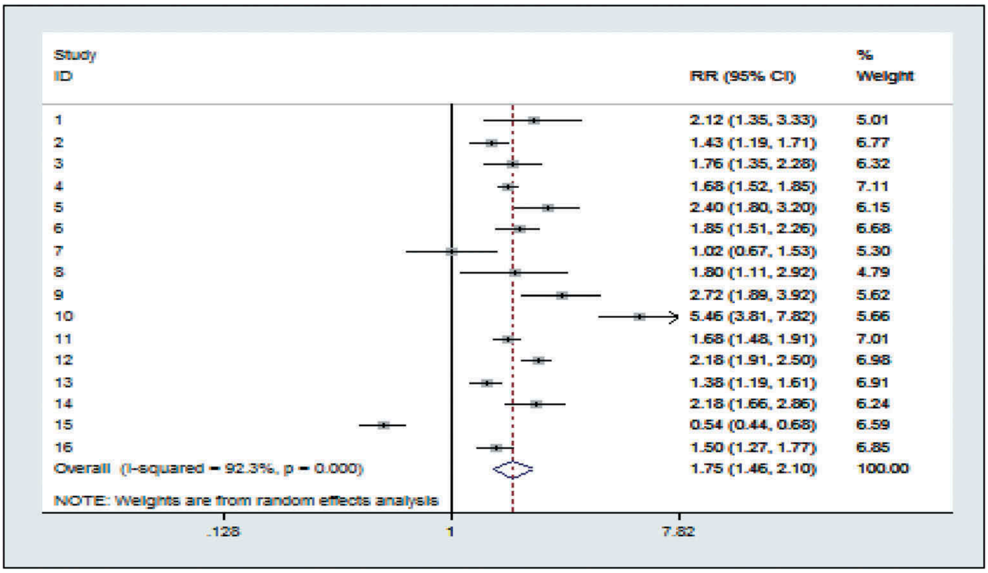

Figure 2. Forest plot of respiratory morbidity. studies (n = 16) have been classified according to the present outcome data in both arms (RR, risk ratio).

There is a large number of new studies on short-term and long-term air pollution exposure, covering a wider geographic area of India. However, through our literature review, we found that direct estimation of the short-term and long-term effects of air pollution in India has rarely been attempted. In India, the health evidence base on AAP is primarily based on cross-sectional or time-series studies which have been conducted in large cities or capital cities through primary as well as secondary data. But still, lack of data remains a challenge in conducting long-term studies on the health impacts of air pollution (particularly ambient) since, in most of the cities, data have no proper interval with a lot of missing data points. It should not be surprising that very large parts of the population in urban areas breathe air that does not meet Indian standards and not follow the WHO Air Quality Guidelines. More than half of the cities in India included in the

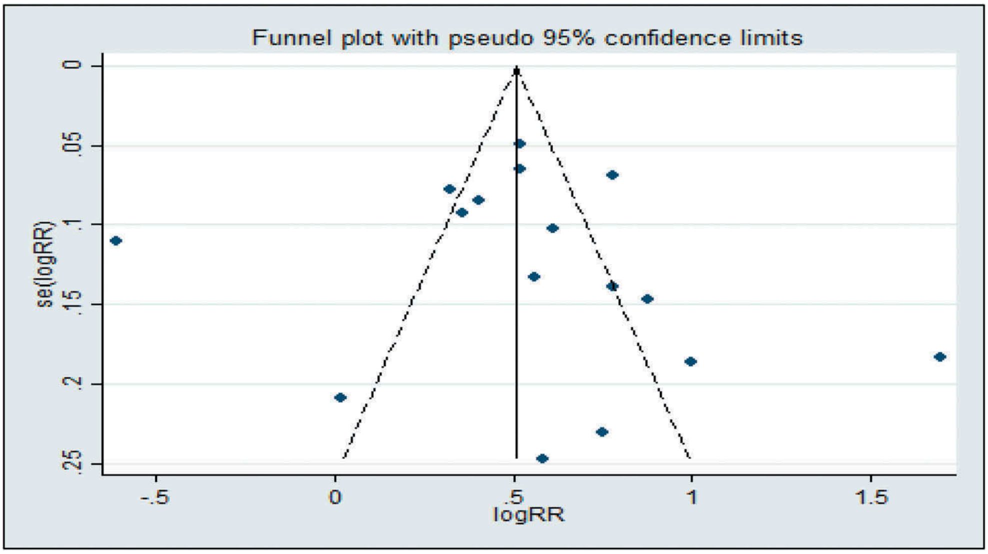

Figure 3. Funnel plot of the 16 studies to examine the relationship between AAP and respiratory morbidity (outcome: response rate).
Note: The vertical line corresponds to the summary log (RR) as estimated from the fixed-effect model (RR, risk ratio)

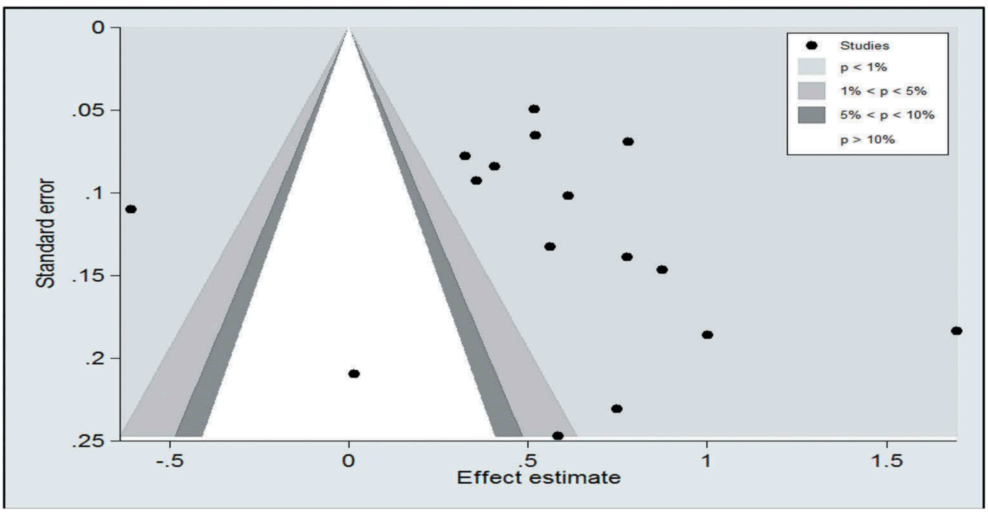

Figure 4. Contour-enhanced funnel plot of the 16 studies to show the significance level of the selected studies on respiratory morbidity.
Note: The vertical line corresponds to the estimated summary log(RR). The shaded areas correspond to levels of statistical significance defined by the p-value of a z-test for the log RR (RR, risk ratio)

National Air Quality Monitoring Program (NAMP), two critical measures, $PM_{2.5}$ and PM_{10} (daily and annual levels), routinely exceed Interim Target-1 levels (75 and 150 μg/m^3 for daily and 35 and 70 μg/m^3 annually, respectively), as designated by the WHO. Higher ambient concentrations of particulate matter ($PM_{2.5}$ and PM_{10}), SPM, O_3 and NO_X are responsible for an excess number of mortality and morbidity in the various megacities of India.

The risk of exposure to AAP occurs in both rural and urban areas. However, the routine monitoring of air quality in India is nearly exclusively confined to urban centers (Gordon et al. 2018), like in cities of Delhi, Mumbai, Kolkata, Chennai, Bengaluru and other states capitals. The

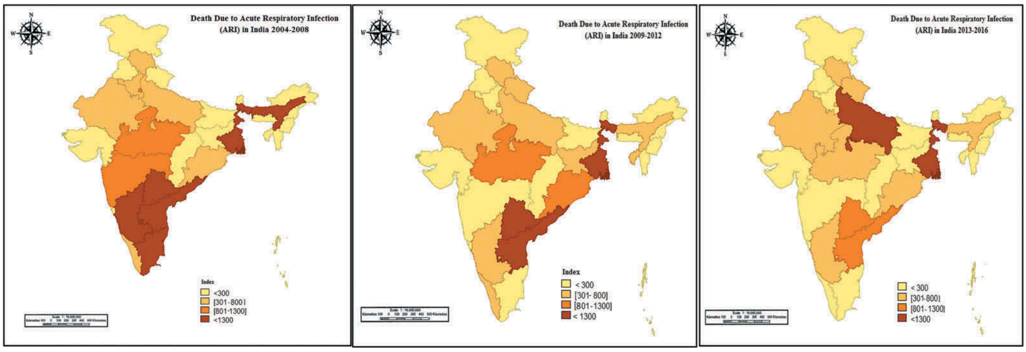

Figure 5. Deaths due to acute respiratory infection (ARI) in India (2004–2016).
Source: Data obtained from Ministry of Health and Family Welfare (MOHFW), Government of India. Map Prepared by Authors.

concentration of PM ($PM_{2.5}$ and PM_{10}) in the air of Delhi is higher than other metro cities of India. Since these cities have high population growth, unplanned urban development, increase in consumption patterns and higher demands for transport, energy and other infrastructure, thereby the quality of air is deteriorating and leading to rising incidences of chronic obstructive pulmonary disease (COPD), cancer, ARI and pollution-related ailments (Pande et al. 2002; Ingle et al. 2005; Ghose 2009; Patankar and Trivedi 2011; Maji et al. 2016). Studies conducted primarily in states like West Bengal, Bihar, Uttar Pradesh, Andhra Pradesh and Telangana suggest a higher risk of mortality due to acute respiratory infection (ARI) [Map 1].

We found very few cohort studies that reported mortality estimates for India in the long term. While there is a need for cohort studies to establish the long-term effects of exposure to air pollution on health. However, we found heterogeneity among studies and their outcome because of variation in numerous factors such as health outcome definition, length of exposure, population, climate of the specific city/states, geography, smoking status, previous respiratory diseases and using different study design and methodology.

Evidence has emerged that prevalence of asthma and chronic diseases are high among children and the elderly (Prasad and Sanyal 2016; Central Pollution Control Board, 2012a; Kumar et al. 2004; Chhabra et al. 2001). Another paper also reported that $PM_{2.5}$ exposure leads to a large number of years of life lost (YLL) in the elderly (aged 65 years and above) (Lin et al. 2018). They are particularly vulnerable to air pollution exposure. But very few studies have assessed long-term exposure of AAP and mortality among elderly. It is still unclear what are the ambient pollutants most damaging to the health of the elderly (Simoni et al. 2015). As a consequence, such vulnerable populations have an increased risk of developing short-term and long-term adverse effects related to AAP and thus need a closer follow-up. Therefore, researcher can directly examine the morbidity and mortality pattern of elderly due to AAP. Also, government should focus to improve air quality level in urban areas to reduce respiratory diseases significantly.

Conclusion and recommendation

Ambient air pollution (AAP) has one of the major impacts on human health, triggering and inducing many diseases leading to high morbidities and mortalities in India. The rapid growth in the industrial, power and transportation sectors combined within urbanization have contributed to the rapid increase in AAP levels in India. If India manages to meet the national air quality standard in all the cities exceeding Indian standard, annual premature death will be reduced. The policy makers and legislators must update all laws and regulations related to air pollutions. People can reduce the detrimental effects of AAP by reducing the time spent on outdoor when AQI is beyond a specified level also wearing masks when it's necessary can be a good protector in this respect (Jiang et al. 2018). An effective environmental protection organization should have enough budgets for administration, research, development, monitoring and full control of the environment including AAP. Awareness generation using educational institutes, mass media and different organizations can be made consciously to make people aware of the consequences of AAP. The pollution control authorities in India urgently need proper policies to elevate ambient air quality in terms of $PM_{2.5}$and PM_{10} level to decrease the burden of diseases due to air pollution. Further research on the health effects of AAP should be very beneficial to the researcher, public health officials, industrialists and the general public.

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Conflicts of interest statement

The author declares no conflict of interest in the study.

Disclosure statement

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