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Burden of Outdoor Air Pollution in Kerala, India—A First Health Risk Assessment at State Level

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Academic Editor: Paul B. Tchounwou

Abstract: Ambient air pollution causes a considerable disease burden, particularly in South Asia. The objective of the study is to test the feasibility of applying the environmental burden of disease method at state level in India and to quantify a first set of disease burden estimates due to ambient air pollution in Kerala. Particulate Matter (PM) was used as an indicator for ambient air pollution. The disease burden was quantified in Years of Life Lost (YLL) for the population (30 + years) living in urban areas of Kerala. Scenario analyses were performed to account for uncertainties in the input parameters. 6108 (confidence interval (95% *CI*): 4150–7791) of 81,636 total natural deaths can be attributed to PM, resulting in 96,359 (95% *CI*: 65,479–122,917) YLLs due to premature mortality (base case scenario, average for 2008–2011). Depending on the underlying assumptions the results vary between 69,582 and 377,195 YLLs. Around half of the total burden is related to cardiovascular deaths. Scenario analyses show that a decrease of 10%

in PM concentrations would save 15,904 (95% *CI*: 11,090–19,806) life years. The results can be used to raise awareness about air quality standards at a local level and to support decision-making processes aiming at cleaner and healthier environments.

Keywords: Air pollution; particulate matter; environmental burden of disease; Years of Life Lost (YLL); Kerala; India

1. Introduction

Air pollution is a well-known risk factor causing human ill-health. It is responsible for thousands of premature deaths, particularly in South Asia [1]. Considering global levels of ambient particulate matter (PM), India ranks tenth and thus is among the most polluted countries with an annual average PM_{10} level of 134 $\mu g/m^3$. 42 Indian cities are listed among the 100 most polluted cities worldwide [2]. According to the latest update of the Global Burden of Disease (GBD) 2010 study 627,426 (95% *CI*: 528,681–726,434) deaths were caused by ambient PM pollution in India. Combining mortality and morbidity effects of air pollution, and using the Disability-Adjusted Life Year (DALY) as a measure for population health, 17,760,000 healthy life years (95% *CI*: 15,201,700–20,705,000) were lost in India in 2010. Most of the DALYs were lost due to mortality effects (95.6%) with only 4.4% attributable to outcomes of morbidity [1].

To estimate the health risk of air pollution an indicator needs to be defined that approximates the level of air pollution. One of the best studied indicators are PM_{10} (coarse particles smaller than 10 μm in aerodynamic diameter) and $PM_{2.5}$ (fine particles smaller than 2.5 μm in aerodynamic diameter) [3,4]. PM is a mixture of small components and thus not a homogeneous stressor and its composition varies by location and sources [5–7]. The respirable fraction of PM consists mainly of organic and elemental carbonaceous materials; inorganic components such as sulfate, nitrate, and ammonium; and metal components such as iron, aluminum, nickel, copper, zinc, and lead. Coarse particles comprise primarily organic and elemental carbon and metals such as silicon, magnesium, iron, ions like sulphates, nitrates, and ammonium [8]. In India the main anthropogenic sources of PM are road traffic emissions, industrial combustion plants, commercial, and residential combustion such as cooking with solid fuels, and agricultural activities [8]. Additional regional sources are road dust, waste burning, and sea salt [9].

Exposure to PM can cause several adverse health effects, including mortality and morbidity outcomes [5]. The exposure to PM is associated with an increased health risk when inhaling fine particles. Once inhaled, these particles can harm the cardiovascular system by inflammation or coagulation. Additionally, the respiratory system can be harmed, because PM can trigger pulmonary oxidative stress [6].

Based on scientific evidence regarding adverse health consequences of air pollution the World Health Organization (WHO) recommends an annual mean of not more than 20 $\mu g/m^3$ and 10 $\mu g/m^3$ for PM_{10} and $PM_{2.5}$, respectively [3]. To prevent and control air pollution, India issued the Air Prevention and Control of Pollution Act in 1981 and developed National Ambient Air Quality Standards (NAAQS) to regulate pollutant emissions. In 2009, the standards were updated and 12 air pollutants are currently regulated. The annual mean standards for PM_{10} and $PM_{2.5}$ are 60 and 40 $\mu g/m^3$ [10].

To tackle the air pollution problem, regional assessments are necessary especially in countries such as India where large health and environmental disparities exist. The differences between states in terms of population, climate, and air pollution are large and need to be considered in state-specific assessments. Such adapted risk assessments can help to raise awareness for ambient air pollution and the resulting health risks. Furthermore, they can support policy and programs and help to introduce measures to reduce ambient air pollution [11–13].

For our purpose, we focus Kerala, a state on the southern tip of the Indian subcontinent with a coastline of about 580 km. In 2011, Kerala had 33.4 million inhabitants (16.0 million males and 17.4 million females). Nearly half of these people reported living in urban areas (47.7%). With a sex ratio of 1084 women per 1,000 men, Kerala has the highest share of females in the population among all Indian states [14].

In this study we aim to test the feasibility of the environmental burden of disease at state level in India. In addition, we quantify a first set of disease burden estimates due to ambient air pollution in urban areas of Kerala.

2. Data and Methods

2.1. Quantification Method

In our study, the disease burden of ambient air pollution was quantified by the mortality component (Years of life Lost) of the DALY. The DALY measure generally combines morbidity (Years Lived with Disability-YLDs) and mortality (Years of Life Lost-YLLs) in one measure and thus allows comparisons of different diseases, interventions, populations, and periods [15,16].

The YLLs were calculated using the Environmental Burden of Disease (EBD) approach, an extension of the burden of disease approach, which was developed by WHO, the World Bank and the Harvard School of Public Health [17,18]. The number of deaths in a certain age-group attributable to ambient PM exposure was multiplied with the remaining life expectancy at the age of death. The deaths attributable to PM exposure were calculated as a population attributable fraction (PAF) and suitable concentration-response functions [17]. The concentration-response functions are available with a confidence interval (CI) and the upper and lower bounds were used to calculate the CI of the YLLs. The PAF was calculated with this formula [19,20]:

$$PAF = \frac{\sum_{i=1}^{n} P_i \left(RR_i - 1 \right)}{\sum_{i}^{n} P_i \left(RR_i - 1 \right) + 1} \tag{1}$$

The calculations were performed in an Excel environment using predefined and adapted spreadsheets as published by the WHO [21]. Uniform age-weights and no time-discount were applied for the estimates. To account for the state-specific setting, the life expectancies from the urban population of Kerala was used [22].

2.2. Data Input

Several datasets are needed to perform the calculation of EBD [17]. Table 1 summarizes the input data used in this study.

Table 1. Input data used for the calculation of EBD due to ambient air pollution in urban Kerala.

Data	Reference area	Source	Reference Year	Stratified by		
				Age	Sex	Rural/ Urban
PM data	Measured data for six cities in Kerala	CPCB [23]	2008–2011	–	–	Urban only
Concentration-response function for PM and all-cause mortality/cardio vascular mortality	Meta-analyses based on studies from the U.S.A., Germany, the Netherlands, Switzerland, Canada, China and New Zealand	Hoek *et al.* [24]	1976–2008 (range of the follow-up period in the meta-analyses)	Applicable only for people aged 30 years and older	–	–
	Four cities in northern China	Zhang *et al.* [25]	1998–2009	Applicable only for people aged 30 years and older	Yes	–
Population data	Kerala	Government of India [14]	2011	Yes (1 year age groups)	Yes	Yes
Life table	Kerala	Registrar General India [22]	2006–2010	Yes (1 year age groups)	Yes	Yes
Cause specific mortality data	Kerala (coverage only 12.2% of total deaths)	Office of the registrar general, India [26]	2010	Yes (10 years age groups)	Yes	–
Mortality data	Kerala (no ICD for cause of death)	Office of the registrar India [27]	2011	Yes (10 years age groups)	Yes	Yes

ICD: International Classification of Diseases, a standard diagnostic tool to classify diseases.

2.2.1. Particulate Matter Data

PM_{10} is measured all over India. It is monitored in an eight-hour sampling twice a week, which results in 104 observations per year [8]. For Kerala, these data were available on the internet (open access) in form of regularly published reports by the Central Pollution Control Board (CPCB) [23]. Table 2 shows the data available for Kerala, which were based on 17 measurement sites in six cities. Therefore only the burden of diesease of the urban population can be assessed.

Table 2. Annual mean PM_{10} concentration (in $\mu g/m^3$) measured by CPCB at six locations in Kerala from 2008 to 2011, Source [23].

City	Number of Stations	2008	2009	2010	2011
Kochi	7	43	42	36	38
Kozhikode	2	34	32	42	46
Thrissur	1	–	–	31	33
Mallapuram	1	–	–	39	30
Trivandrum	4	67	61	56	58
Kollam	2	–	–	47	53

For the quantification of the disease burden attributable to air pollution an annual mean value of 44.9 $\mu g/m^3$ PM_{10} was calculated by taking into account all measured values from 2008 to 2011 to smooth out annual outliers. The most current evidence on concentration-response functions relate to $PM_{2.5}$ instead of PM_{10} but for Kerala comprehensive $PM_{2.5}$ data were not available, therefore, PM_{10} measurements were converted into $PM_{2.5}$ by using recommendations of the WHO [3] and two recent studies from India [23,28] suggesting $PM_{2.5}$ to PM_{10} ratios of 0.4, 0.5, and 0.7.

Furthermore, a counterfactual value was needed to assess the negative health effects above the comparative exposure concentration. For $PM_{2.5}$ so far no threshold was identified below which no negative health effects of $PM_{2.5}$ occur [3,29]. Nevertheless zero pollution is not a realistic assumption due to natural sources of $PM_{2.5}$. Therefore we used two different counterfactual levels: (a) a theoretical minimum exposure of 7.3 $\mu g/m^3$ derived from the largest cohort study on air pollution in the United States of America [29–31]; and (b) the guideline value of 10 $\mu g/m^3$ as recommended by the WHO air quality guidelines [3].

2.2.2. Concentration-Response Functions

Two concentration-response functions were used in this assessment. One is from an international meta-analysis because it was assumed that pooled results of different studies increases the consistency and validity of the concentration-response function. The other is from a Chinese cohort study because the exposure situation in China is rather comparable with the one in India (Table 3). For clear assignment of concentration-response functions and health data, International Classification of Disease 10 (ICD-10) codes were used.

Table 3. Selected concentration-response functions for all-cause mortality and cardiovascular mortality and $PM_{2.5}/PM_{10}$ exposure.

Source	All-Cause Mortality (ICD-10: A00-R99)	Cardiovascular mortality (ICD-10: I00-I99)	Unit
Hoek *et al.* [24]	1.062 (95% *CI*: 1.04–1.083)	1.11 (95% *CI*: 1.05–1.16)	per 10 $\mu g/m^3$ change in $PM_{2.5}$
Zhang *et al.* [25]	1.24 (95% *CI*: 1.22–1.27)	1.23 (95% *CI*: 1.19–1.26)	per 10 $\mu g/m^3$ change in PM_{10}

2.2.3. Population Data

Population data were obtained from the Indian census 2011 [32]. The data are stratified by states, sex, five-year age groups, and the rural-urban status. Life tables for Kerala with reference years from 2006 to 2011 are also available from the census [22].

2.2.4. Mortality Data

For mortality data, two data sources were used. For the EBD calculation only natural deaths (ICD 10: A00-R99) and for the scenario analysis only cardiovascular deaths (ICD 10: I00-I99) are needed. Therefore data from the Report on Medical Certification of Cause of Death 2010 [26] and the Vital statistics of India based on the Civil Registration System 2011 [27] were combined by applying the cause of death rates to the total number of deaths. These data are stratified by states, sex, five-year age groups) and urban-rural status.

Table 4 shows the demographic data for the urban population of Kerala in the year 2010. From the about 33.4 million inhabitants 7,610,740 men and 8,307,037 women lived in urban areas. The largest numbers of individuals are in the age group 0 to 14 years (676,030 boys and 647,412 girls). A sex difference is visible with 696,297 more women in the total population. The opposite distribution can be found in the mortality data. More men than women died in 2010 (48,292 men and 33,346 women). Most of the deaths occurred in older age groups (age group 70+:5727 per 100,000 men and 3,872 per 100,000 women, respectively).

Table 4. Demographic data of the population living in urban areas of Kerala in 2010, stratified by age groups and sex, Sources: [14,22,26,27].

Age	Population		Natural Deaths		Natural Deaths per 100,000 People		Cardiovascular Deaths		Cardiovascular Deaths per 100,000 People	
	M	W	M	W	M	W	M	W	M	W
<1	116,460	113,490	1,679	1,286	1442	1133	41	36	35	32
1–4	477,265	459,537	227	173	48	38	22	10	5	2
5–9	616,113	590,795	133	105	22	18	14	13	2	2
10–14	676,020	647,412	146	115	22	18	15	15	2	2
15–19	632,095	612,644	395	232	62	38	75	29	12	5
20–24	618,543	661,205	386	250	62	38	73	32	12	05
25–29	564,939	670,117	680	355	120	53	128	88	23	13
30–34	535,243	641,998	644	340	120	53	121	84	23	13
35–39	553,478	681,566	1,296	658	234	97	304	145	55	21
40–44	539,207	627,153	1,262	606	234	97	296	134	55	21
45–49	527,161	593,487	3,291	1,338	624	225	853	300	162	51
50–54	446,274	480,709	2,786	1,084	624	225	722	243	162	51
55–59	414,667	424,358	5,633	2,423	1,359	571	1,666	712	402	168
60–64	333,759	355,965	4,534	2,032	1,359	571	1,341	597	402	168
65–69	218,695	258,174	5,679	3,438	2,597	1,332	1,871	1,346	856	521
70+	340,821	488,427	19,519	18,911	5,727	3,872	7,185	7,784	2,108	1,594
Total	7,610,740	8,307,037	48,290	33,346	635	401	14,727	11,569	194	139

2.2.5. Scenario Analyses

To reflect existing uncertainties in the input data, several input parameters were altered: two concentration-response functions, three $PM_{2.5}$ to PM_{10} ratios, and two counterfactual values were used to estimate the impact of parameter changes on the EBD estimates. Combining the different options resulted in ten scenarios (Table 5). The baseline scenario (*Natural Deaths (ND)_Baseline (1)*) summarizes the assumption of a conservative concentration-response function, the mid value of the $PM_{2.5}$ to PM_{10} ratio, and the theoretical minimum risk exposure as counterfactual value.

Table 5. Parameter scenario descriptions by considered concentration-response functions, $PM_{2.5}$ to PM_{10} ratios, and counterfactual values. ND: Natural Deaths, CD: Cardiovascular Deaths.

Scenario	Concentration-Response Function (per 10 µg/m³)	$PM_{2.5}$ to PM_{10} Ratio	Counterfactual Value in µg/m³
Natural deaths excluding accidents (ICD 10: A00–R99)			
ND_Baseline (1)	1.062 (95% *CI*: 1.040–1.083) [a]	0.5 [c]	7.3 [e]
ND_Low $PM_{2.5}$ to PM_{10} ratio (2)	1.062 (95% *CI*: 1.040–1.083) [a]	0.4 [d]	7.3 [e]
ND_High $PM_{2.5}$ to PM_{10} ratio (3)	1.062 (95% *CI*: 1.040–1.083) [a]	0.7 [d]	7.3 [e]
ND_Alternative counterfactual value (4)	1.062 (95% *CI*: 1.040–1.083) [a]	0.5 [c]	10 [c]
ND_Alternative CRF (5)	1.24 (95% *CI*: 1.22–1.27) [b]	-	20 [c]
Deaths caused by diseases of the circulatory system (ICD 10: I00–I99)			
CD_Baseline (6)	1.11 (95% *CI*: 1.050–1.16) [a]	0.5 [c]	7.3 [e]
CD_Low $PM_{2.5}$ to PM_{10} ratio (7)	1.11 (95% *CI*: 1.050–1.16) [a]	0.4 [d]	7.3 [e]
CD_High $PM_{2.5}$ to PM_{10} ratio (8)	1.11 (95% *CI*: 1.050–1.16) [a]	0.7 [d]	7.3 [e]
CD_Alternative counterfactual value (9)	1.11 (95% *CI*: 1.050–1.16) [a]	0.5 [c]	10 [c]
CD_Alternative CRF (10)	1.23 (95% *CI*: 1.19–1.26) [b]	–	20 [c]

[a] Hoek *et al.* [24], [b] Zhang *et al.* [25], [c] WHO [3], [d] Satsangi *et al.* [28], [e] Lim *et al.* [31].

Air pollution is not a constant environmental factor and the counterfactual values used for the quantification are currently not achievable in India, therefore two additional and more realistic assumptions were considered: a possible decrease and increase in $PM_{2.5}$ by 10% each (Table 6).

Table 6. Air pollution scenario descriptions by considered concentration-response functions, $PM_{2.5}$ to PM_{10} ratios, counterfactual values, and assumptions on the development of PM.

Scenario	Concentration-Response Function (per 10 µg/m³)	$PM_{2.5}$ to PM_{10} Ratio	Counterfactual Value in µg/m³	Assumption ($PM_{2.5}$ Development)
Natural deaths ICD 10: A00-R99				
ND_10% increase in $PM_{2.5}$ (11)	1.062 (95% *CI*: 1.040–1.083) [a]	0.5 [c]	7.3 [e]	10% less $PM_{2.5}$
ND_10% decrease in $PM_{2.5}$ (12)	1.062 (95% *CI*: 1.040–1.083) [a]	0.5 [c]	7.3 [e]	10% more $PM_{2.5}$

Table 6. *Cont.*

Scenario	Concentration-Response Function (per 10 μg/m³)	PM$_{2.5}$ to PM$_{10}$ Ratio	Counterfactual Value in μg/m³	Assumption (PM$_{2.5}$ Development)
Deaths caused by diseases of the circulatory system ICD 10 I00-I99				
CD_10% increase in PM$_{2.5}$ (13)	1.11 (95% *CI*: 1.050–1.16) [a]	0.5 [c]	7.3[e]	10% less PM$_{2.5}$
CD_10% decrease in PM$_{2.5}$ (14)	1.11 (95% *CI*: 1.050–1.16) [a]	0.5 [c]	7.3[e]	10% more PM$_{2.5}$

[a] Hoek *et al.* [24], [c] WHO [3], [e] Lim *et al.* [31].

3. Results and Discussion

3.1. Results

In the recent years (2008–2011), the annual mean PM$_{10}$ concentrations in ambient air in urban areas of Kerala did not exceed the national guideline value of 60 μg/m³—except for two values in Trivandrum in 2008 and 2009 which were slightly above the guideline value (Table 2). However, the measured values were considerably higher than the guidelines recommended by WHO (20 μg/m³ PM$_{10}$).

In the baseline scenario (*ND_Baseline (1)*), 6,108 (*CI*: 4150–7791) of the 81,636 total natural deaths in the urban population of Kerala can be attributed to ambient air pollution by PM$_{2.5}$ (Figure 1). Hence 7.5% of deaths can be attributed to PM$_{2.5}$. Stratified by sex and in absolute numbers, more attributable deaths were modeled for men with 3,613 (*CI*: 2455–4609) deaths, as compared to 2495 (*CI*: 1695–3183) deaths for women. According to the assumptions in the different scenarios the results vary markedly. The lowest burden in terms of premature deaths was estimated for scenario *ND_High PM$_{2.5}$ to PM$_{10}$ ratio (3)*, using the more conservative value for the conversion factor from PM$_{2.5}$ to PM$_{10}$ (0.4). The highest burden can be found in scenario *ND_Alternative CRF (5)*, with 22,785 (*CI*: 21,912–23,909) attributable deaths.
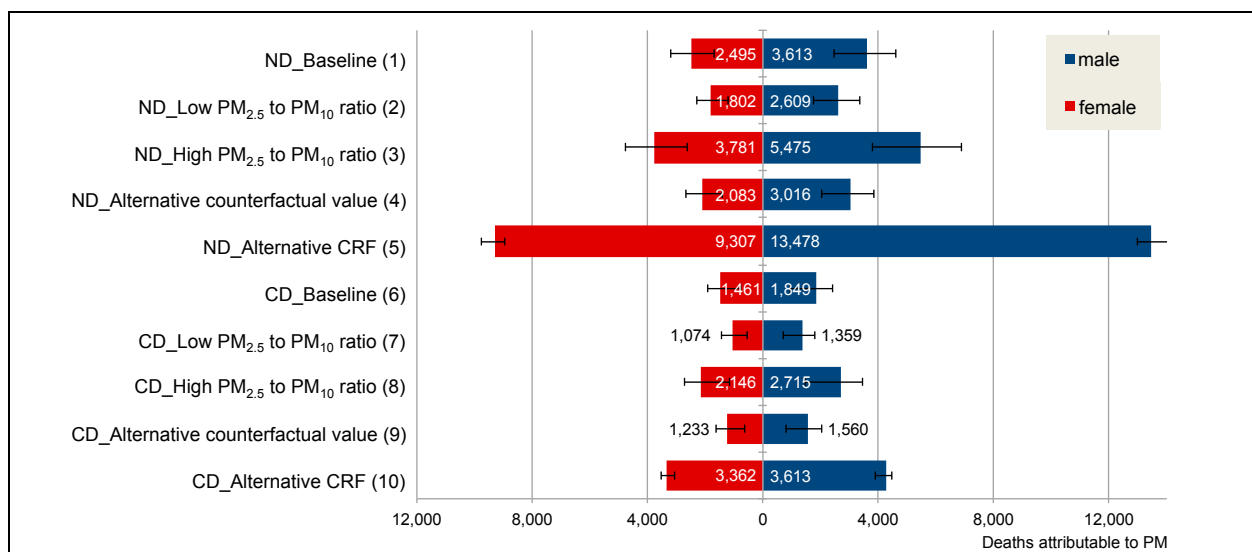

Figure 1. Deaths attributable to air pollution (PM) by different scenarios for the male and female urban population of Kerala. ND: Natural Deaths, CD: Cardiovascular Deaths.

The scenario analysis shows that many deaths which can be attributed to ambient air pollution by $PM_{2.5}$ are due to cardiovascular causes. In the baseline scenario (*Cardiovascular Deaths (CD)_Baseline (6)*) 51% of the male and 49% of the female cardiovascular deaths can be attributed to air pollution. The sex difference was smaller than for the total natural deaths. The lowest number of premature death cases occur in scenario *CD_Low PM₂.₅ to PM₁₀ ratio (7)* and the highest in scenario *CD_Alternative CRF (10)*.

Comparable patterns of disease burden were estimated for YLLs attributable to ambient $PM_{2.5}$ (Table 7). In the baseline scenario (*ND_Baseline (1)*) for total natural deaths, 96,359 (*CI*: 65,479–122,917) life years were lost due to $PM_{2.5}$. The absolute burden was higher in the male population with 58,868 (*CI*: 40,003–75,094) YLLs compared to 37,490 (*CI*: 25,476–47,823) YLLs in the female population. Per 100,000 people 605 (*CI*: 411–772) years of life are lost. Scenario *ND_Low PM₂.₅ to PM₁₀ ratio (2)* and *ND_High PM₂.₅ to PM₁₀ ratio (3)* show the impact of the change in the conversion factor from $PM_{2.5}$ to PM_{10}. If a conversion factor of 0.4 was applied, the burden was calculated to 69,582 (*CI*: 46,873–89,463) YLLs. If the ratio was 0.7, the burden more than doubles to 146,020 (*CI*: 100,860–183,589) YLLs. Scenario *ND_Alternative counterfactual value (4)* represents the results of using a counterfactual value of 10 $\mu g/m^3$ $PM_{2.5}$, with 80,434 (*CI*: 54,375–103,085) YLLs. These results are lower compared to the baseline scenario because adverse health effects below 10 $\mu g/m^3$ $PM_{2.5}$ were not included. The highest burden was estimated for scenario *ND_Alternative CRF (5)* with 359,465 (*CI*: 345,695–377,195) YLLs, which is more than 3.5 times that of the baseline scenario.

Table 7. YLLs and YLLs per 100,000 inhabitants due to $PM_{2.5}$ in urban areas of Kerala, stratified by sex, *CI* in parentheses.

Scenario	YLLs			YLLs per 100,000		
	Men	Women	Total	Men	Women	Total
ND_Baseline	58,868	37,490	96,358	773	451	605
(1)	(40,003–75,094)	(25,476–47,823)	(65,479–122,917)	(526-987)	(307–576)	(411–772)
ND_Low PM₂.₅	42,510	27,072	69,582	559	326	437
to PM₁₀ ratio (2)	(28,636–54,656)	(18,237–34,807)	(46,873–89,463)	(376–718)	(220–419)	(294–562)
ND_High PM₂.₅	89,208	56,812	146,020	1172	684	917
to PM₁₀ ratio (3)	(61,619–112,160)	(39,242–71,429)	(100,861–183,589)	(810–1,474)	(472–860)	(634–1,153)
ND_Alternative counterfactual value (4)	49,139	31,294	80,433	646	377	505
	(33,219–62,977)	(21,156–40,107)	(54,375-103,084)	(436–827)	(25–483)	(342–648)
ND_Alternative CRF (5)	219,608	139,857	359,465	2,885 (2775–3028)	1684 (1619–1767)	2258 (2172–2370)
	(211,195–230,440)	(134,500–146,755)	(345,695–377,195)			
CD_Baseline	28,086	19,880	47,966	369	239	301
(6)	(14,637–36,706)	(10,361–25982)	(24,998–62,688)	(192–482)	(125–313)	(157–394)
CD_Low PM₂.₅	20,639	14,609	35,248	271	176	221
to PM₁₀ ratio (7)	(10,520–27,4717)	(7,447–19,407)	(17,367–46,824)	(138–360)	(90–234)	(113–294)
CD_High PM₂.₅	41,235	29,188	70,423	542	351	442
to PM₁₀ratio (8)	(22,376–52,394)	(15,839–37,087)	(38,215–89,481)	(294–688)	(191–446)	(240–562)

Table 7. *Cont.*

Scenario	YLLs			YLLs per 100,000		
	Men	Women	Total	Men	Women	Total
CD_Alternative counterfactual value (9)	23,688 (12,184–31,257)	16,768 (8,624–22,125)	40,456 (20,808–53,382)	311 (160–411)	202 (104–266)	254 (131–335)
CD_ Alternative CRF (10)	64,608 (58,899–68,061)	45,732 (41,691–48,176)	110,340 (100,590–116,237)	849 (774–849)	551 (502–580)	693 (632–730)

In scenario analyses, which specifically estimate the disease burden for cardiovascular diseases in the baseline scenario (*CD_Baseline (6)*) 47,966 (*CI*: 24,998–62,688) years of life are lost due to ambient PM$_{2.5}$ pollution. As in the natural deaths calculation, a sex difference is visible with more male YLLs.

The age pattern of the disease burden in the baseline scenario (*ND_Baseline (1)*) is shown in Figure 2. For both sexes the disease burden is increasing throughout the age-groups, with some minor decreases. The highest burden in natural deaths is in the oldest age group, with over 27% of YLLs in men and even 42% of YLLs in women. The cardiovascular death burden is also highest in the oldest age group.
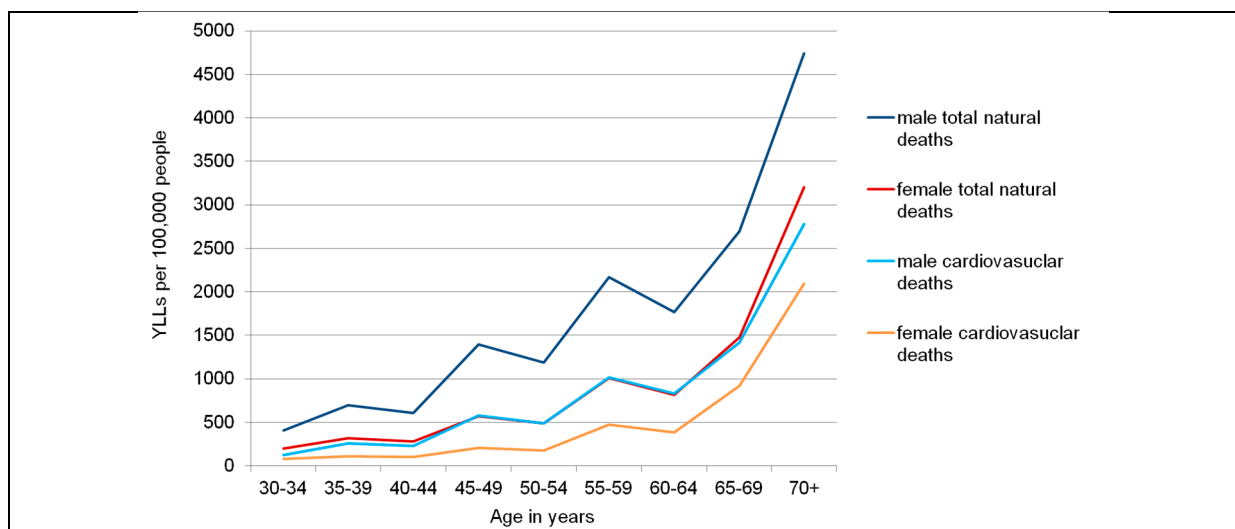

Figure 2. Age patterns of YLLs per 100,000 people due to PM in the baseline scenarios (*ND_Baseline (1) and CD_Baseline (6)*), in urban Kerala.

Assuming that air pollution and thus the PM$_{2.5}$ concentration levels will change in the future, Figure 3 shows the impact of a 10% decrease of PM$_{2.5}$ (*ND_10% decrease in PM$_{2.5}$ (12)* and *CD_10% decrease in PM$_{2.5}$ (14)*) and a 10% increase *ND_10% increase in PM$_{2.5}$ (11)* and *CD_10% increase in PM$_{2.5}$ (13)*). Improved air quality regarding PM$_{2.5}$ would reduce the burden by 15,904 (*CI*: 11,090–19,806) to 80,455 (*CI*: 54,389–103,111) YLLs as compared to the baseline scenario (*ND_Baseline (1)*). In scenario *CD_10% increase in PM$_{2.5}$ (13)*, 41,745 (*CI*: 21,519–54,992) YLLs still can be attributed to air pollution by PM$_{2.5}$, which would be 5954 (*CI*: 3479–7696) YLLs less compared to the cardiovascular baseline scenario (*CD_Baseline (6)*).

A worsening of air quality by 10% more PM$_{2.5}$ would increase the burden to 109,242 (*CI*: 74,547–13,826) YLLs. In total 12,884 (*CI*: 9068–15,909) YLLs more would occur due to higher PM$_{2.5}$ concentrations. The cardiovascular burden would increase to 53,930 (*CI*: 28,405–69,951) YLLs.
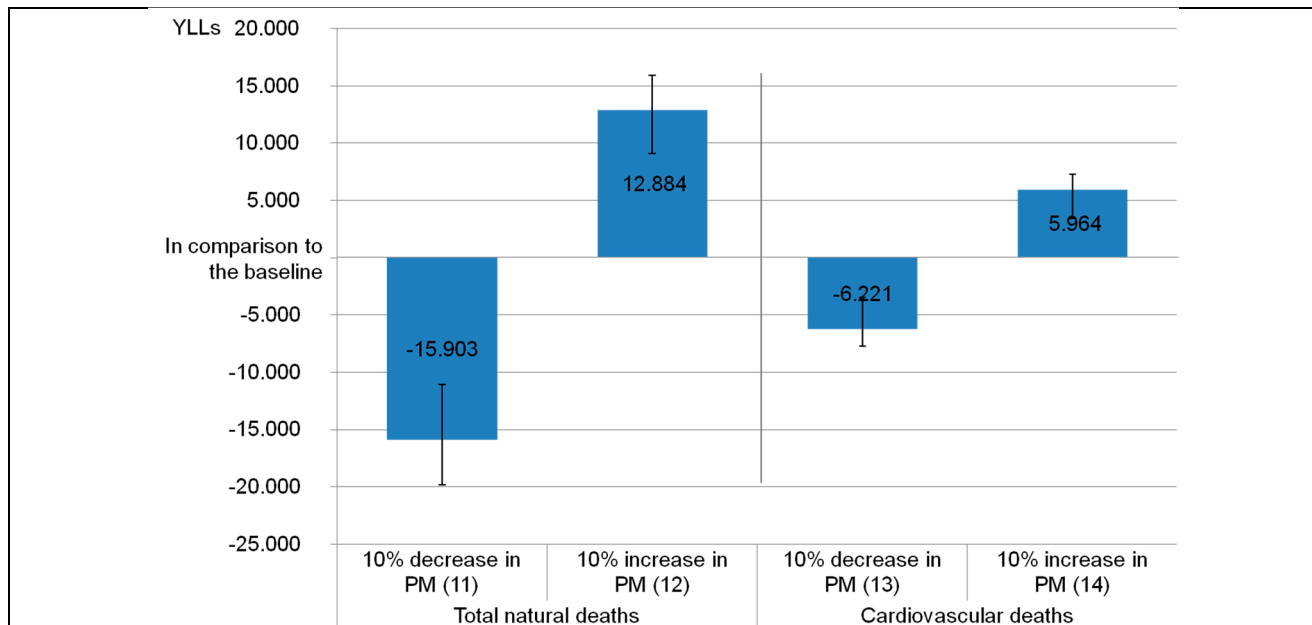
Figure 3. Impact on the burden of disease in urban Kerala of 10% less and 10% more PM2.5 compared to the baseline scenario, scenarios 11 to 14.

3.2. Discussion

The aim of the study was to test the feasibility of the environmental burden of disease approach at state level in Kerala, India, and to quantify a first set of disease burden estimates due to ambient air pollution by PM2.5. In general, despite some limitations in data availability, the method was applicable at state level. The disease burden due to ambient air pollution for the urban population was estimated to the best of our knowledge for the first time, using state specific data such as PM10 concentrations, population and mortality data. Data on air pollution were freely available, but the locations of the single measurement sites were missing as well as exposure data for the rural population. Therefore, population-weighted exposure modeling was not possible. Nevertheless, the results show the importance of air pollution as a threat to population health in Kerala.

The ambient PM10 values in Kerala did not exceed the Indian national guidelines. However, these standards are lagging far behind other national and international standards [9]. Our results support this criticism by showing the burden of PM2.5. Further, realistic future scenarios of PM2.5 were assessed, demonstrating that a worsening of air pollution (a 10% increase in PM2.5) would increase the mortality-associated disease burden by around 13%. By improving air quality (a 10% decrease in PM2.5), around 17% of the disease burden attributed to PM2.5 could be prevented.

The scenario analysis shows that around half of the natural deaths which can be attributed to PM2.5 are due to cardiovascular causes (*ND_Baseline (1)* and *CD_Baseline (6)*): natural deaths 6108 (*CI*: 4150–7791) and cardiovascular deaths 3,311 (*CI*: 1725–4327) attributable to air pollution). The other 50% of the natural deaths may have other causes like lung cancer, chronic obstructive pulmonary disease, or other respiratory diseases.

In our assessment, we used air pollution data published by CPCB of India. Comparing these data to other sources provides some differences in the EBD results. The national annual average of PM10 concentration in ambient air in India from 2009 to 2012 was 132 μg/m³ [33]. This value is much

higher than the value we used for our calculations (44 µg/m^3 PM$_{10}$). In the last GBD study a weighted annual mean PM$_{2.5}$ of 27.2 µg/m3 was used to quantify the burden of air pollution in India [34,35], which is in the range of PM$_{2.5}$ we applied (18.0 to 31.5 µg/m^3 PM$_{2.5}$). Data from the same source extracted for Kerala provide an even lower value of 14.5 µg/m^3 PM$_{2.5}$. This population weighted mean refers to the state of Kerala in total and thus includes rural areas [35].

In the scenario analyses different PM$_{2.5}$ to PM$_{10}$ ratios were assessed because so far PM$_{2.5}$ is not comprehensively measured in India and no agreed and exact ratio is available. The ratios applied in our study vary from 0.4 to 0.7 [23,28]. The results differ accordingly: when applying a ratio of 0.4, the burden is 69,582 (*CI*: 46,873–89,463) YLLs for natural causes, which is around half of the burden when applying a ratio of 0.7 (146,020 (*CI*: 100,860–183,589)).

The disease burden estimates are a first set of results and should be interpreted with caution. No estimates for Kerala were available so far, therefore estimates from India and South Asia were used to compare the results. In the recent burden of disease estimates published by WHO, the premature deaths attributable to ambient air pollution in South East Asia were 52 per 100,000 persons for 2012 [36]. In our study, 38 natural deaths per 100,000 people (*CI*: 26–49) can be attributed to ambient air pollution in Kerala. However, considering the much broader reference area in the WHO estimates, the differences are reasonable—particularly so as the WHO applied much higher PM level values than we did in our assessment.

In the GBD 2010 study the mortality part of disease burden of ambient PM in India was estimated to be 1,358 (95% *CI*: 1192–1617) YLLs per 100,000 people, which is much higher than our estimate (605 (*CI*: 411–772) YLLs per 100,000 people). Reasons for the large differences could be the different input data. In the GBD 2010 study, many more deaths were considered in the calculation (896 male and 771 female deaths per 100,000 for India and our numbers are 635 males and 401 females deaths per 100,000 for Kerala) and different concentration-response functions and life expectancies were applied because in the GBD study an international comparison was targeted [31]. Another reason for the comparably low disease burden calculated in the present assessment could be that nearly half of the population in Kerala was living in urban areas, but only around 34.4% of deaths were reported there.

Because of the complex and data-demanding calculations, our study also faces some limitations, mostly related to data quantity and quality. The best available data were used, but still limitations and uncertainties occur which are discussed in the following.

No comprehensive data on the cause of death were available. Therefore, data from the Report on Medical Certification of Cause of Death 2010 and the Vital statistics of India based on the Civil Registration System 2011 were combined, while keeping in mind that the combination of two data sources can lead to several uncertainties. Data on causes of death were classified in 10-year age groups. In order to enable reliable quantifications it was necessary to distribute these data to five-year age groups using population data. This does not give an accurate distribution of the death causes. Nevertheless, for the assignment of the remaining life expectancy to the age groups and the quantification of the YLLs it is sufficiently detailed.

The highest available age group in the mortality data is 70 years and older. In this age group the disease burden caused by PM is the highest for natural deaths as well as for cardiovascular deaths with respect to absolute numbers of premature deaths. More detailed data for the age groups older than 70 years would allow more accurate results because there is evidence that elderly people are more

susceptible to the effects of air pollution [37]. Additionally, young children, undernourished people, and those with pre-existing health problems should be considered in more detail because they would benefit most from better air quality [38,39].

Beside the uncertainties related to the PM exposure, the conversion factor from PM_{10} to $PM_{2.5}$ and mortality data, the applicability of concentration-response functions is questionable. Over the last decades a growing number of experimental and epidemiological studies increased the knowledge of the association between PM exposure (especially $PM_{2.5}$) and adverse health effects [3,4,24,40], but evidence concerning the statistical relationship, which is needed for an health risk assessment, is still limited, especially for India [13]. During the last few years, two time series studies for Delhi and Chennai were conducted to assess the link between PM exposure in ambient air and the number of natural deaths. However, these results cannot be directly used in this assessment, because the study focused on short-term exposure solely [41]. So far no cohort studies on long term exposure to air pollution and mortality have been reported for India [42]. Therefore, and because of broad consistency of Asian time-series studies with European and North American studies, the Health Effects Institute (HEI) supports the use of results from Western cohort studies, if data for estimating the burden of disease attributable to air pollution in Asia is missing [42].

Nevertheless, this approach has limitations because the concentration-response functions were derived at lower levels of air pollution than observed in Asia and thus the results must be interpreted with caution. Therefore, two concentration-response functions were used in this assessment to show the impact of this input variable. Comparing scenario *ND_Alternative counterfactual value* with an excess risk of 6% (95% *CI*: 4%–8%) per 10 μg/m^3 increase in $PM_{2.5}$ exposure with scenario *ND_Alternative CRF* and a 24% (95% *CI*: 22%–27%) excess risk per 10 μg/m^3 PM_{10} increase, shows a difference of 279,031 (*CI*: 291,321–274,110) YLLs. Thus, applying the concentration-response function estimated for China resulted in a burden four times as high as when using the pooled concentration-response function. These large differences prove the strong influence of the concentration-response function on the EBD calculation and the need to research on concentration-response functions for India and other South Asian countries. Compared to the other input variables the choice of the concentration-response function has the largest effect on the results.

Several other adverse health effects of exposure to PM are discussed, but convincing evidence is still lacking [4,40]. As soon as sufficient evidence is available, further disease endpoints need to be included in the estimation processes to better illustrate and underline the importance of air pollution as a major health threat. Additionally the related health data (mortality and morbidity) is needed to assess the effects of PM comprehensively. If, for example, prevalence data on cardiovascular and respiratory health outcomes would be available, the morbidity part (years lived with disability) could be quantified as well.

The approach presented here can be adopted by other Indian states by applying respective population and ambient air pollution data. However, the availability of concentration-response functions should be examined because, compared to other Indian states, the air pollution levels in Kerala are relatively low. Thus if necessary, an adapted concentration-response function for higher air pollution levels and another slope (e.g. supralinear) should be applied to avoid an overestimation [43]. Finally, the assessment needs to be further developed in the direction of an integrated approach by including rural settings as well as indoor exposure as suggested by Balakrishnan, Dhaliwal and Shah [12].

3.2.1. Implication for Further Research

- Conduct a cohort study to assess the effects of long-term air pollution exposure on health outcomes (mortality and morbidity) and to derive representative concentration-response functions for Indian settings.
- Expand the number of measurement parameters of air pollution, like $PM_{2.5}$, to provide more specific and reliable data for health risk assessments. Likewise, the number of measurement sites should be increased to also cover rural areas. This would allow a much more comprehensive risk assessment.
- Assess indoor air pollution as well and include measurements in the YLL estimations at state level.

3.2.2. Practical Implications

The identification of the sources of air pollution is another step to develop effective mitigation policies. India's development over the last decades is characterized by a social and economic progress which is directly linked to industrialization, urbanization, and motorization, all leading to an increase in ambient air pollution. This development will most probably continue in the future [44]. In particular, the demand for personal transport and the amount of goods transportation are increasing steadily [45]. This in turn leads to an increase of pollutant emissions from vehicle exhausts. Therefore, actions to reduce hazardous emissions are needed, such as fuel emission standards or a shift to a safer and cleaner public transport alternative [9]. Because public transport is not an option for everybody, actions addressing the road conditions and the traffic itself are needed. Poor road conditions, the high number of vehicles, waterlogging during monsoons, and people on the street interrupt the traffic and lead to traffic congestions, which in turn can increase the fuel consumption [44]. Better road maintenance, paving of unpaved roads, and silt removal would be possible actions. The low quality of fuel and lubricating oil currently used also contributes to poor air quality [44]. CPCB is already discussing a road map for fuel quality improvement in India.

Further pollution sources are solid fuels used for cooking and industrial emissions. The latter need to be regulated by appropriate policies, for example by a shift from coal based industry to the use of cleaner and renewable fuels such as wind or solar energy. A shift to cleaner fuel is also needed for cooking, because indoor air pollution causes a considered health burden [12].

4. Conclusions

Our findings show that the EBD method is applicable at state level and can be applied to other Indian states as well. The results indicate, that even if local air quality standards are met, a considerable health burden for the population living in urban Kerala can be assumed, which can be partly prevented by taking actions to reduce air pollution. Compared to other Indian states Kerala shows relative low annual PM levels, thus the burden of disease due to PM in other Indian states is expected to be even higher. Further estimates for other Indian states can help to complete the overall picture and allow for state-wise comparisons.

Author Contributions

Myriam Tobollik conceived the study, built the models, gathered and compiled the data and performed the analyses. Myriam Tobollik drafted the first version of the manuscript with editorial input from Dietrich Plass. Oliver Razum, Dirk Wintermeyer, Dietrich Plass interpreted the results and provided critical feedback on the manuscript. Oliver Razum helped setting up the study. All authors read and approved the final version of the manuscript.

Conflicts of Interest

The authors declare no conflict of interest. The founding sponsors had no role in the design of the study; in the collection, analyses, or interpretation of data; in the writing of the manuscript, and in the decision to publish the results.

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