Annexure: Summary of studies obtained by conducting a structured literature review

Note

i. The studies listed below are the result of a structured review conducted by the author using relevant keywords. However, please note that this list may not be exhaustive.
ii. The studies illustrated in the table are not subjected to study quality evaluation.
iii. The studies contained in the annexure exclusively concern $PM_{2.5}$ exposure, provided as an illustrative example.

Table 1: Studies pertaining to exposure to ambient $PM_{2.5}$ and all-cause mortality

All- cause mortality					
S. No	Year of study	Location	Pollutant	Remarks	Reference
1	2010 - 2016	Delhi	$PM_{2.5}$	A stronger association was observed between $PM_{2.5}$ levels and mortality in Delhi, which was stronger among older adults. For a 10 µg/m^3 increase in $PM_{2.5}$ level, non-accidental mortality increased by 0.31%.	[1]
2	2009 - 2016	Indo-Gangetic plain (IGP)	Aerosols (Black carbon (BC), $PM_{2.5}$, and PM_{10}) and Trace gases (NO_2, SO_2, and O_3)	The single pollutant model of $PM_{2.5}$ showed a significant impact on mortality. A considerable increase in mortality was observed when $PM_{2.5}$ levels were >60 µg/m^3. Mortality was higher during hazy days.	[2]
3	2012	Six regions of India	$PM_{2.5}$	$PM_{2.5}$ pollution causes 1.1 million premature deaths in India, with 80% linked to anthropogenic emissions. Reduction in emissions in the IGP and Central India regions can lower mortality across other parts of the country.	[3]
4	2016	29 cities	$PM_{2.5}$	The premature mortality burden attributable to $PM_{2.5}$ exposure in these cities was 1,14,700 (1,04,100–1,25,500) deaths. There is an urgent need for stricter $PM_{2.5}$ standards because the current supra-linear $PM_{2.5}$ exposure-response relationship offers limited effectiveness in reducing mortality.	[4]

5	2015	India	$PM_{2.5}$	$PM_{2.5}$ pollution causes 1.09 (0.93–1.25) million deaths in a year. Non-linear exposure-response functions indicate that significant reductions in the burden of pollution in India will require substantial decreases in $PM_{2.5}$ levels.	[5]
6	2001-2015	Varanasi	$PM_{2.5}$	Exposure to $PM_{2.5}$ leads to 5700 (2800-7500) annual premature deaths (0.16% of the population). Meeting the WHO guideline value, the annual premature mortality burden will be reduced by 92%.	[6]
7	2013 - 2017	Delhi	$PM_{2.5}$	There is a 0.52% rise in non-trauma all-cause mortality for each 10 $\mu g/m^3$ increase in short-term $PM_{2.5}$ exposure. Notably, this effect is two-fold greater during winter compared to summer and has a more pronounced impact on males (0.57%) than on females (0.52%).	[7]
8	2016	Punjab	$PM_{2.5}$	2,582 deaths linked to $PM_{2.5}$ exposure, with preventable deaths ranging from 246 (WHO guideline: 10 $\mu g/m^3$) to 159 (Indian NAAQS: 40 $\mu g/m^3$).	[8]
9	2013–2016	Delhi	$PM_{2.5}$ chemical species	Significant associations were observed between non-trauma all-cause mortality and both $PM_{2.5}$ (2.65% increase in mortality per Inter-quartile region (IQR) total $PM_{2.5}$ mass) and specific chemical components. Notably, NO_3^-, NH_4NO_3, Cr, NH_4^+, EC, and OC subspecies exhibited a more pronounced impact on mortality compared to the overall $PM_{2.5}$ mass concentration.	[9]
10	2017	31 non-attainment cities	$PM_{2.5}$	Total $PM_{2.5}$ attributable premature mortality cases was 80,447 deaths. IHD was the leading cause of death.	[10]

Table 2: Studies pertaining to exposure to ambient $PM_{2.5}$ and cardiovascular diseases

Cardiovascular disease							
S. No	Year of Study	Location	Pollutant / Parameter	Type of study / sample information	Outcome Variable	Remarks	Reference
1	2001	India and Saudi Arabia	$PM_{2.5}$	Long-term exposure study; n = 1,37,809 adults; age group: 35-70	Blood pressure (BP) and hypertension	Chronic exposures to outdoor $PM_{2.5}$ was associated with increased BP and hypertension. $PM_{2.5}$ exposure showed increased odds ratio (OR) of 1.04 for hypertension, per 10 µg/m^3 increase in $PM_{2.5}$.	[11]
2	2010-2016	Delhi	$PM_{2.5}$	Short-term and long-term exposure cohort study; n = 5,342	BP and hypertension	A significant association exists between $PM_{2.5}$ levels and both Systolic blood pressure (SBP) and hypertension, with a more prominent effect observed in individuals with higher waist-hip ratios. Higher average SBP of 1.77 mmHg and 3.33 mmHg per IQR differences in monthly and annual exposures, respectively. The IQR differences in long-term exposures of 1, 1.5, and 2 years increased the risk of incident hypertension by 1.53, 1.59, and 1.16, respectively.	[12]

3	2010-2012	Andhra Pradesh	$PM_{2.5}$, BC	Long-term exposure; cross-sectional; n = 5,531 (Children and parents' study)	BP and hypertension	Among women, a 1 µg/m³ rise in $PM_{2.5}$ corresponded to a 1.4 mmHg increase in SBP, a 0.87 mmHg increase in Diastolic blood pressure (DBP), and a 4% greater likelihood of hypertension. Conversely, no such association was observed among men, indicating a gender-specific link between ambient $PM_{2.5}$ and BP as well as hypertension in women.	[13]
4	2011	Delhi	PM_{10}, SO_X, NO_X, $PM_{2.5}$	n = 2,218; age group: 21–65	Hypertension, blood cell morphological changes	The prevalence of hypertension was nearly 4-times higher in Delhi when compared to the control region.	[14]
5	2010-2019	Japan, South Korea, Thailand, China, Sri Lanka, India, & Nepal	Household $PM_{2.5}$, ground-level O_3, ambient $PM_{2.5}$	Global burden of disease data	Ischemic Heart Disease (IHD) mortality data	Exposure to household $PM_{2.5}$ and ambient $PM_{2.5}$ has a considerable influence on IHD mortality, especially in the case of the elderly population in India.	[15]
6	2010-2012	Andhra Pradesh	$PM_{2.5}$	n = 3,278	Carotid intima-media thickness (CIMT)	Exposure to $PM_{2.5}$ was associated with CIMT (1.79% increase in CIMT for each additional 1 µg/m³ increase in $PM_{2.5}$) among men from a population with a high prevalence of cardiometabolic risk factors.	[16]
7	2007-2010	China, Ghana, India, Mexico, Russia and South Africa	$PM_{2.5}$	Long-term exposure-cohort study; n = 45,625	Stroke	6.55% of the stroke cases could be attributable to ambient $PM_{2.5}$ in the study population. OR= 1.13 for each 10 µg/m³ increase in $PM_{2.5}$.	[17]

8	2010-2012	Peri-urban South India	$PM_{2.5}$ and BC	n = 3,017	CIMT, carotid-femoral pulse wave velocity (cf-PWV) and augmentation Index (AIx).	Exposure to PM in a peri-urban population in South India was linked to vascular damage, and this damage exhibited gender-specific effects. These differences may be attributed to variations in the sources of personal exposure. A 10 $\mu g/m^3$ increase of $PM_{2.5}$ was positively associated with CIMT (0.026 mm), cf-PWV (0.069 m/s) and AIx (0.8%) among men.	[18]
9	2003-2018	21 high-, middle, and low-income countries including India	$PM_{2.5}$	Prospective cohort study; n=1,57,436 adults; age group: 35–70	Cardiovascular disease events, mortality, and other non-accidental mortality	An increment of 10 $\mu g/m^3$ in $PM_{2.5}$ was linked to a heightened risk of cardiovascular disease events (HR = 1.05), myocardial infarction (HR = 1.03), stroke (HR = 1.07), and cardiovascular disease mortality (HR = 1.03). These results exhibited a similarity to countries with high $PM_{2.5}$ concentrations (>35 $\mu g/m^3$).	[19]

Table 3: Studies pertaining to exposure to ambient $PM_{2.5}$ and respiratory diseases

Respiratory disease							
S. No	Year of study	Location	Pollutant / Parameter	Type of study / sample information	Health outcome	Remarks	Reference
1	2008 - 2010.	Delhi	$PM_{2.5}$, PM_{1}	n = 378 outdoor exercisers	Spirometry test	The outdoor exercisers are exposed to high PM levels and they are at high risk of lung function impairment due to the deposition of PM in the airways.	[20]
2	February 2017 - August 2017	Chennai	NO_2, SO_2, CO and $PM_{2.5}$	Case-control Study; n = 1000; age group: 5 – 75.	Allergic symptoms	Showed a clear association between exposure to $PM_{2.5}$ with the prevalence of dyspnea and eosinophilia among the subjects of the survey. The OR=7.4 signifies the likelihood of experiencing dyspnea in those with allergies present compared to those without allergies.	[21]
3	August 2013- November 2013	Maharashtra	$PM_{2.5}$	Cross-sectional study; n = 2400 adults	Spirometry test	A significant association was observed between exposure to $PM_{2.5}$ with respiratory symptoms among adults and the effect was higher among the residents near an industrial area.	[22]

4	2005-2006	Chennai, Delhi, Hyderabad, Kolkata, Mumbai, Indore, Meerut, and Nagpur	SO_2, NO_2, $PM_{2.5}$, and PM_{10}	n = 4,665 children under age 5.	Cough without a fever, Cough with a fever and fever without a cough	There are statistically significant increases in the likelihood of a cough for a child exposed to higher levels of $PM_{2.5}$ or PM_{10} but not for exposure to NO_2. As the $PM_{2.5}$ level increases by one standard deviation above the mean (119 µg/m³) the likelihood of cough increases by 6.01 probability points.	[23]

Table 4: Studies pertaining to exposure to ambient $PM_{2.5}$ and reproductive & birth defects

Reproductive and birth defects							
S. No	Year of study	Location	Pollutant / Parameter	Type of study / sample information	Health outcome	Remarks	Reference
1	2015–2016	India	$PM_{2.5}$	Observational study; n = 2,18,152 children	Height-for-age	Early-life exposure to $PM_{2.5}$ is associated with child height deficits. 100 µg/m³ increase in $PM_{2.5}$ in the month of birth was associated with a 0.05 standard deviation reduction in child height	[24]
2	1998–2016	India, Pakistan, and Bangladesh	$PM_{2.5}$	Case-control Study; n = 34,197 mothers	Pregnancy loss (i.e., stillbirth and miscarriage).	A strong association was established between exposure to $PM_{2.5}$ and pregnancy loss. A 10 µg/m³ increment in $PM_{2.5}$ was associated with an OR for pregnancy loss of 1·03.	[25]

3	2015–16	India	$PM_{2.5}$	Cross-sectional study; n = 1,49,416	LBW	Exposure to $PM_{2.5}$ levels exceeding about 50 µg/m³ during pregnancy is linked to LBW. Children exposed to $PM_{2.5}$ levels exceeding 45 µg/m³ in utero have increased odds of LBW, and the likelihood of LBW rises as $PM_{2.5}$ levels increase, peaking at approximately 70 µg/m³ before levelling off.	[26]
4	2001–2006	Multi-national (24 countries in Africa, Latin America, and Asia)	$PM_{2.5}$	n > 2,90,000 women in 373 institutions	Preterm birth and LBW	On a global scale, there was a correlation between LBW and outdoor $PM_{2.5}$ levels, but not with preterm birth. The OR for LBW was 1.22 when comparing the fourth quartile of $PM_{2.5}$ (> 20.2 µg/m³) to the first quartile (< 6.3 µg/m³). In India, there was an inverse relationship between $PM_{2.5}$ levels and both preterm birth and LBW.	[27]
5	2015–2016	India	$PM_{2.5}$	Retrospective Cohort Study; n=74,47,724; age group: <5 (born between 2009–2016)	All-cause child mortality	Child mortality adjusted hazard ratios were 1.023 and 1.013 per 10 µg/m³ increase of *in utero* and post-delivery lifetime $PM_{2.5}$ exposure.	[28]

Table 5: Studies pertaining to exposure to ambient $PM_{2.5}$ and endocrine diseases

Endocrine disorder							
S. No	Year of study	Location	Pollutant / Parameter	Type of study/sample information	Health outcome	Remarks	Reference
1	2016	Multi-national	$PM_{2.5}$	Longitudinal cohort; n = 1,729,108	Risk of incident diabetes	India has the highest DALYs for diabetes among the top ten most populous countries. The diabetes risk significantly rises above 2.4 µg/m³ of $PM_{2.5}$ and increases moderately at levels above 10 µg/m³. The global impact of $PM_{2.5}$-related diabetes is significant, even at levels considered safe by USEPA and WHO.	[29]
2	2010-2012	28 peri-urban villages in South India	$PM_{2.5}$ and BC	Cross-sectional data; n = 5,065 adults	Blood glucose and diabetic status	No link found between exposure to $PM_{2.5}$/BC and blood glucose or prediabetes/ diabetes prevalence.	[30]
3	2011	Bikaner district	PM_{10}, $PM_{2.5}$ and NO_2	Cross-sectional analysis; n = 3,457; age group: 30 – 70.	Fasting blood sugar and HbA1C levels	Long-term air pollution exposure was associated with impaired fasting glucose, impaired glucose tolerance, and prevalence of diabetes mellitus. The prevalence stood at 8.93%, with a mean HbA1C of 8.67. Concurrently, the levels of PM_{10}, NO_2, and $PM_{2.5}$ were recorded at 156.12 µg/m³, 5.43 µg/m³, and 25.36 µg/m³, respectively.	[31]

Table 6: Studies pertaining to exposure to ambient $PM_{2.5}$ and blood disorders

S. No	Year of study	Location	Pollutant / Parameter	Type of study / sample information	Health outcome	Remarks	Reference
1	2007-2016	India	$PM_{2.5}$	n= 6,40,557; Age group: women of reproductive age (15–49)	Anaemia prevalence	For every 10 µg/m^3 increase in $PM_{2.5}$ exposure, the average anaemia prevalence among Indian women of reproductive age increases by 7.23%.	[32]
2	2015-2016	India	$PM_{2.5}$ and Aerosol optical depth	n=98,557 children (individual analysis) and n = 638 districts (ecological analysis).	Haemoglobin concentration	The district-level ecological analysis found that, for every 10 µg/m^3 increase in ambient $PM_{2.5}$ exposure, average anaemia prevalence increased by 1.90% and average haemoglobin decreased by 0.07g/dL. At the individual level, for every 10 µg/m^3 increase in ambient $PM_{2.5}$ exposure, average haemoglobin decreased by 0.14g/dL.	[33]

Table 7: Studies pertaining to exposure to household air pollution (HAP)

S. No	Year of study	Location	Pollutant / Parameter	Type of study / sample information	Health outcome	Remarks	Reference
1	2010 - 2015	Tamil Nadu	$PM_{2.5}$	Cohort study; n = 1285 pregnant women in the first trimester of pregnancy.	Birth weight	Exposure to $PM_{2.5}$ is associated with LBW. A 10-$\mu g/m^3$ increase in $PM_{2.5}$ exposures was associated with a 4 g decrease in birthweight and 2% increase in prevalence of LBW.	[34]
2	May 2018	Tamil Nadu	$PM_{2.5}$, BC, CO	Cross-sectional study; n = 799 Women; 9- and 20-weeks of gestation.	Gestational blood pressure	There was no significant association observed between $PM_{2.5}$ levels and gestational BP within the study group. However, an increase of 1-log $\mu g/m^3$ in CO exposure was linked to a rise of 0.36 mmHg in DBP.	[35]
3	N/A	Rural South India	$PM_{2.5}$, CO, Lung deposited surface area (LDSA)	Cross-sectional cohort study; 96 households (The mother and one child aged 8–14 years were the participants from each household)	Forced vital capacity (FVC) and forced expiratory volume (FEV) in 1 second, Peak Expiratory Flow Rate (PEFR) and mid expiratory volume.	Children living in households with higher measured LDSA (OR: 1.69) and CO (OR: 1.34) exposure were associated with an increased risk of subnormal lung function. However, there was no significant association with $PM_{2.5}$.	[36]

4	2004-2009	Delhi	$PM_{2.5}$, Solid fuel	Cross-sectional study; n = 15,573	EFV1, FVC and Tuberculosis (TB) prevalence	Duration of solid fuel exposure showed a significant association with active risk of TB. The long-term ambient $PM_{2.5}$ exposure did not show a significant association with the history of active TB.	[37]
5	2018-2021	Multi-national (rural Guatemala, India, Peru, and Rwanda)	$PM_{2.5}$, BC and CO	Cross-sectional analysis n=418 women; age group: 40–79 years	BP	In older women, higher $PM_{2.5}$ exposure correlated positively with SBP and Pulse Pressure (PP). The most significant BP changes were seen between 149 µg/m^3 and 139 µg/m^3, resulting in a 0.53 mmHg increase in SBP and a 0.43 mmHg increase in PP for those aged 65.	[38]
6	2018 - 2020	Multi-national (rural Guatemala, India, Peru, and Rwanda)	$PM_{2.5}$, BC and CO	N=3200 Pregnant women	Birth weight	Prenatal exposure to $PM_{2.5}$ and BC was linked to LBW. 7·3 µg/m^3 and 74·51 µg/m^3 reduction in prenatal BC and $PM_{2.5}$ exposure was associated with an increase in birthweight of about 22 g and 14.8 g, respectively.	[39]

Bibliography

[1] B. Krishna, S. Mandal, K. Madhipatla, K. S. Reddy, D. Prabhakaran, and J. D. Schwartz, "Daily nonaccidental mortality associated with short-Term PM2.5exposures in Delhi, India," *Environ. Epidemiol.*, 2021, doi: 10.1097/EE9.0000000000000167.

[2] N. Singh, A. Mhawish, T. Banerjee, S. Ghosh, R. S. Singh, and R. K. Mall, "Association of aerosols, trace gases and black carbon with mortality in an urban pollution hotspot over central Indo-Gangetic Plain," *Atmos. Environ.*, vol. 246, Feb. 2021, doi: 10.1016/j.atmosenv.2020.118088.

[3] Liji M. David, A. R. Ravishankara, John K. Kodros, Jeffrey R. Pierce, Chandra Venkataraman, and Pankaj Sadavarte, "Premature Mortality Due to PM2.5 Over India:Effect of Atmospheric Transport and Anthropogenic Emissions," *GeoHealth* , vol. 3, pp. 2–10, May 2019, doi: 10.1029/2018GH000169.

[4] P. Saini and M. Sharma, "Cause and Age-specific premature mortality attributable to PM2.5 Exposure: An analysis for Million-Plus Indian cities," *Sci. Total Environ.*, vol. 710, Mar. 2020, doi: 10.1016/j.scitotenv.2019.135230.

[5] A. J. Cohen *et al.*, "Estimates and 25-year trends of the global burden of disease attributable to ambient air pollution: an analysis of data from the Global Burden of Diseases Study 2015," *Lancet*, vol. 389, no. 10082, pp. 1907–1918, May 2017, doi: 10.1016/S0140-6736(17)30505-6.

[6] V. Jain, S. Dey, and S. Chowdhury, "Ambient PM2.5 exposure and premature mortality burden in the holy city Varanasi, India," *Environ. Pollut.*, vol. 226, pp. 182–189, 2017, doi: 10.1016/j.envpol.2017.04.028.

[7] P. Joshi *et al.*, "Impact of acute exposure to ambient PM2.5 on non-trauma all-cause mortality in the megacity Delhi," *Atmos. Environ.*, vol. 259, no. June, p. 118548, 2021, doi: 10.1016/j.atmosenv.2021.118548.

[8] K. Ravindra, T. Singh, and S. Mor, "Preventable mortality attributable to exposure to air pollution at the rural district of Punjab, India," *Environ. Sci. Pollut. Res.*, vol. 29, no. 21, pp. 32271–32278, 2022, doi: 10.1007/s11356-022-19668-z.

[9] P. Joshi, S. Dey, S. Ghosh, S. Jain, and S. K. Sharma, "Association between Acute Exposure to PM2.5Chemical Species and Mortality in Megacity Delhi, India," *Environ. Sci. Technol.*, vol. 56, no. 11, pp. 7275–7287, 2022, doi: 10.1021/acs.est.1c06864.

[10] M. Nair, H. Bherwani, S. Mirza, S. Anjum, and R. Kumar, "Valuing burden of premature mortality attributable to air pollution in major million-plus non-attainment cities of India," *Sci. Rep.*, vol. 11, no. 1, Dec. 2021, doi: 10.1038/s41598-021-02232-z.

[11] R. E. Arku *et al.*, "Long-term exposure to outdoor and household air pollution and blood pressure in the Prospective Urban and Rural Epidemiological (PURE)

study," *Environ. Pollut.*, vol. 262, Jul. 2020, doi: 10.1016/j.envpol.2020.114197.

[12] D. Prabhakaran *et al.*, "Exposure to particulate matter is associated with elevated blood pressure and incident hypertension in urban India," *Hypertension*, pp. 1289–1298, 2020, doi: 10.1161/HYPERTENSIONAHA.120.15373.

[13] A. Curto *et al.*, "Ambient Particulate Air Pollution and Blood Pressure in Peri-urban India," *Epidemiology*, vol. 30, no. 4, pp. 492–500, Jul. 2019, doi: 10.1097/EDE.0000000000001014.

[14] M. Banerjee *et al.*, "Hematological, immunological, and cardiovascular changes in individuals residing in a polluted city of India: A study in Delhi," *Int. J. Hyg. Environ. Health*, vol. 215, no. 3, pp. 306–311, Apr. 2012, doi: 10.1016/j.ijheh.2011.08.003.

[15] A. Mumtaz, N. Rehman, A. Haider, and S. Rehman, "Long-Term Air Pollution Exposure and Ischemic Heart Disease Mortality Among Elderly in High Aging Asian Economies," *Front. Public Heal.*, vol. 9, Feb. 2022, doi: 10.3389/fpubh.2021.819123.

[16] O. T. Ranzani *et al.*, "Association between ambient and household air pollution with carotid intima-media thickness in peri-urban South India: CHAI-Project," *Int. J. Epidemiol.*, vol. 49, no. 1, pp. 69–79, Feb. 2020, doi: 10.1093/ije/dyz208.

[17] H. Lin *et al.*, "Ambient PM2.5 and Stroke: Effect Modifiers and Population Attributable Risk in Six Low- and Middle-Income Countries," *Stroke*, vol. 48, no. 5, pp. 1191–1197, May 2017, doi: 10.1161/STROKEAHA.116.015739.

[18] O. T. Ranzani *et al.*, "Personal exposure to particulate air pollution and vascular damage in peri-urban South India," *Environ. Int.*, vol. 139, Jun. 2020, doi: 10.1016/j.envint.2020.105734.

[19] P. Hystad *et al.*, "Associations of outdoor fine particulate air pollution and cardiovascular disease in 157 436 individuals from 21 high-income, middle-income, and low-income countries (PURE): a prospective cohort study," *Lancet Planet. Heal.*, vol. 4, no. 6, pp. e235–e245, 2020, doi: 10.1016/S2542-5196(20)30103-0.

[20] C. N. Kesavachandran, R. Kamal, V. Bihari, M. K. Pathak, and A. Singh, "Particulate matter in ambient air and its association with alterations in lung functions and respiratory health problems among outdoor exercisers in national capital region, India," *Atmos. Pollut. Res.*, vol. 6, no. 4, pp. 618–625, Jul. 2015, doi: 10.5094/APR.2015.070.

[21] M. Ammasi Krishnan *et al.*, "Effects of ambient air pollution on respiratory and eye illness in population living in Kodungaiyur, Chennai," *Atmos. Environ.*, vol. 203, pp. 166–171, Apr. 2019, doi: 10.1016/j.atmosenv.2019.02.013.

[22] U. Gawande, A. Khanvilkar, S. Kadam, and G. Salvitthal, "Effects of ambient air pollution on respiratory health of adults: findings from a cross-sectional study

in Chandrapur, Maharashtra, India," *Int. J. Res. Med. Sci.*, pp. 1546–1557, 2016, doi: 10.18203/2320-6012.ijrms20161226.

[23] A. Ghosh and A. Mukherji, "Air pollution and respiratory ailments among children in urban India: Exploring causality," *Economic Development and Cultural Change*, vol. 63, no. 3. University of Chicago Press, pp. 191–222, Oct. 01, 2015, doi: 10.1086/677754.

[24] D. Spears, S. Dey, S. Chowdhury, N. Scovronick, S. Vyas, and J. Apte, "The association of early-life exposure to ambient PM2.5 and later-childhood height-for-age in India: An observational study," *Environ. Heal. A Glob. Access Sci. Source*, vol. 18, no. 1, Jul. 2019, doi: 10.1186/s12940-019-0501-7.

[25] T. Xue, T. Guan, G. Geng, Q. Zhang, Y. Zhao, and T. Zhu, "Estimation of pregnancy losses attributable to exposure to ambient fine particles in south Asia: an epidemiological case-control study," *Lancet Planet. Heal.*, vol. 5, no. 1, pp. e15–e24, Jan. 2021, doi: 10.1016/S2542-5196(20)30268-0.

[26] N. Goyal and D. Canning, "The association of in-utero exposure to ambient fine particulate air pollution with low birth weight in India," *Environ. Res. Lett.*, vol. 16, no. 5, May 2021, doi: 10.1088/1748-9326/abf18e.

[27] N. L. Fleischer *et al.*, "Outdoor air pollution, preterm birth, and low birth weight: Analysis of the world health organization global survey on maternal and perinatal health," *Environ. Health Perspect.*, vol. 122, no. 4, pp. 425–430, 2014, doi: 10.1289/ehp.1306837.

[28] J. Liao *et al.*, "Child Survival and Early Lifetime Exposures to Ambient Fine Particulate Matter in India: A Retrospective Cohort Study," *Environ. Health Perspect.*, vol. 130, no. 1, Jan. 2022, doi: 10.1289/EHP8910.

[29] B. Bowe, Y. Xie, T. Li, Y. Yan, H. Xian, and Z. Al-Aly, "The 2016 global and national burden of diabetes mellitus attributable to PM 2·5 air pollution," *Lancet Planet. Heal.*, vol. 2, no. 7, pp. e301–e312, Jul. 2018, doi: 10.1016/S2542-5196(18)30140-2.

[30] A. Curto *et al.*, "Lack of association between particulate air pollution and blood glucose levels and diabetic status in peri-urban India," *Environ. Int.*, vol. 131, Oct. 2019, doi: 10.1016/j.envint.2019.105033.

[31] M. Gupta, R. P. Agrawal, B. L. Meena, Ramesh, J. K. Meel, and R. Agrawal, "Association between Long Term Exposure to Air Pollution, Impaired Fasting Glucose, Impaired Glucose Tolerance and Prevalence of Diabetes.," *J. Assoc. Physicians India*, vol. 70, no. 4, pp. 11–12, Apr. 2022.

[32] E. Chaudhary *et al.*, "Reducing the burden of anaemia in Indian women of reproductive age with clean-air targets," *Nat. Sustain.*, Aug. 2022, doi: 10.1038/s41893-022-00944-2.

[33] U. Mehta, S. Dey, S. Chowdhury, S. Ghosh, J. E. Hart, and A. Kurpad, "The Association between Ambient PM2.5 Exposure and Anemia Outcomes among Children under Five Years of Age in India," *Environ. Epidemiol.*, vol. 5, no. 1, Feb. 2021, doi: 10.1097/EE9.0000000000000125.

[34] K. Balakrishnan *et al.*, "Exposures to fine particulate matter (PM2.5) and birthweight in a rural-urban, mother-child cohort in Tamil Nadu, India," *Environ. Res.*, vol. 161, pp. 524–531, Feb. 2018, doi: 10.1016/j.envres.2017.11.050.

[35] W. Ye *et al.*, "Association between personal exposure to household air pollution and gestational blood pressure among women using solid cooking fuels in rural Tamil Nadu, India," *Environ. Res.*, vol. 208, May 2022, doi: 10.1016/j.envres.2022.112756.

[36] S. Patel *et al.*, "Associations between household air pollution and reduced lung function in women and children in rural southern India," *J. Appl. Toxicol.*, vol. 38, no. 11, pp. 1405–1415, Nov. 2018, doi: 10.1002/jat.3659.

[37] V. Patel *et al.*, "Long-term exposure to indoor air pollution and risk of tuberculosis," *Indoor Air*, vol. 31, no. 3, pp. 628–638, May 2021, doi: 10.1111/ina.12756.

[38] L. Nicolaou *et al.*, "Cross-sectional analysis of the association between personal exposure to household air pollution and blood pressure in adult women: Evidence from the multi-country Household Air Pollution Intervention Network (HAPIN) trial," *Environ. Res.*, vol. 214, no. August, 2022, doi: 10.1016/j.envres.2022.114121.

[39] K. Balakrishnan *et al.*, "Exposure–response relationships for personal exposure to fine particulate matter (PM2·5), carbon monoxide, and black carbon and birthweight: an observational analysis of the multicountry Household Air Pollution Intervention Network (HAPIN) trial," *Lancet Planet. Heal.*, vol. 7, no. 5, pp. e387–e396, 2023, doi: 10.1016/S2542-5196(23)00052-9.