atmosphere

Supplementary

Traffic-related Particulate Matter and Cardiometabolic Syndrome: A Review

C. M. Sabbir Ahmed, Huanhuan Jiang, Jin Y. Chen and Ying-Hsuan Lin

Table S1: Results of databases with subsequent keywords "Traffic related air pollution"—"Particulate matter"—"Human health"—"Metabolic syndrome", grouped by various time intervals.

Search database with keywords	**Time interval**				
	1980-1990	**1991-2000**	**2001-2010**	**2011-2018**	**Total**
Google Scholar					
Traffic related air pollution	9	299	4490	12800	17598
Particulate matter	1	103	2840	8870	11814
Human health	1	48	1270	4040	5359
Metabolic syndrome	0	0	39	205	**244**
Web of Science					
Traffic related air pollution	0	28	285	586	899
Particulate matter	0	5	73	273	351
Human health	0	0	2	11	13
Metabolic syndrome	0	0	1	2	**3**
PubMed					
Traffic related air pollution	0	15	180	499	694
Particulate matter	0	2	59	212	273
Human health	0	0	0	3	3
Metabolic syndrome	0	0	0	0	**0**
JSTOR					
Traffic related air pollution	1	18	268	168	455
Particulate matter	0	7	200	122	329
Human health	0	1	53	26	80
Metabolic syndrome	0	0	0	4	**4**

Table S2: Summary of selected 25 articles on traffic-related PM and their impacts in the progression of cardiometabolic syndromes.

Type of study	Reference	Study period	Exposure dose/concentration	Study subject	Gender/Age	Major health outcomes
Epidemiological	Riediker, 2007 [1]	2001	In-vehicle $PM_{2.5}$: an average of 24 μg/m^3	Human	Highway patrol troopers/23-30 years	In-vehicle exposure to $PM_{2.5}$ may cause pathophysiologic changes that involve inflammation, coagulation, and cardiac rhythm.
Epidemiological	Alexeeff et al., 2018 [2]	2010-2015	BC: 0.36 μg/m^3, NO: 4.9 ppb and NO_2: 9.9 ppb	Human	Adults	Street-level differences in long-term exposure to traffic-related air pollutants were associated with higher risk of cardiovascular events among the elderly, indicating that within-neighborhood differences in TRAP are important to cardiovascular health. Associations among the general population were consistent with results found in previous studies, though not statistically significant.
Epidemiological	Riediker et al., 2018 [3]	N/A	$PM_{2.5}$ at work: 88.48 μg/m^3, $PM_{2.5}$ at home: 48.17 μg/m^3, and $PM_{2.5}$ at night: 21.5μg/m^3	Human	Male Swiss highway maintenance workers	Found reduced modulation of HRV with the exposure of $PM_{2.5}$.
Epidemiological	Mazidi & Speakman, 2018 [4]	CVD: 2011-2013; Obesity: since 1984	$PM_{2.5}$: 11.9±1.7 μg/m3, ozone: 0.06±0.01 ppm	Human	Female and male/age>35 years for cardiovascular disease study	There was a spatial association between $PM_{2.5}$ exposure and the leading causes of death and disability in United States. The effect of $PM_{2.5}$ was considerably greater in areas where obesity is more prevalent. Hypertension is a possible mediator of the association of $PM_{2.5}$ and both CVD and stroke.
Controlled human exposure	Peretz et al., 2008 [5]	N/A	Diesel exhaust associated fine PM: 100 or 200 μg/m^3	Human	Male and female/18–49 years	Short-term exposure to DE is associated with acute endothelial response and vasoconstriction of a conductance artery.
Epidemiological	Wu et al., 2011 [6]	2008-2009	$PM_{2.5}$ and CO in car	Human	Nonsmoking, young, and healthy taxi drivers	Results support the association of exposure to traffic-related air pollution with altered cardiac autonomic function in young healthy adults free of cardiovascular compromises. Negative associations of HRV with $PM_{2.5}$ were observed.
Epidemiological	Gan et al., 2011 [7]	1994-2002	BC: (1.49 ± 1.10) × 10^{-5}/m, $PM_{2.5}$: 4.08 ± 1.63 μg/m^3, NO_2: 32.1 ± 8.0 μg/m^3 and NO: 32.0 ± 11.9 μg/m^3	Human	45–85 years	There was a clear linear exposure–response relationship between BC and coronary events (hospitalization and mortality).
Epidemiological	Hou et al., 2012 [8]	N/A	Personal $PM_{2.5}$: average 94.6 μg/m^3 (office workers), average 126.8 μg /m^3 (truck drivers); Personal EC: average 13.1 μg /m^3 (office workers), average 17.3 μg /m^3 (truck drivers); ambient PM_{10}: average 116.7 μg /m^3 (office workers), average 123.5 μg /m^3 (truck drivers)	Human	Male and female/average 30.27 years (office workers), average 33.5 years (truck drivers)	Short-term exposure to ambient PM is associated with increased blood telomere length, consistent with telomere roles during acute inflammatory responses. Longer exposure may shorten telomere as expected after prolonged pro-oxidant exposures. The observed telomere alterations may participate in the biological pathways of short- and long-term PM effects.
Epidemiological	Steenhof et al., 2014 [9]	2009	PM_{10}: 76 (18–450) μg/m^3, $PM_{2.5}$: 39 (8–167) μg/m^3, EC, OC, Cu, Ni, V, NO_2: 20 (9–34) ppb	Human	N/A	PM was associated with changing in total WBC counts, number of neutrophils, monocytes and, inflammatory response.
Toxicological	Wagner et al., 2014 [10]	2011	$PM_{2.5}$: 356 ± 87 μg/m^3, O_3: 0.485 ± 0.042 ppm from highway and industry site	Sprague-Dawley rat	Male/8 weeks	Cardiovascular depression like heart rate, HRV, and blood pressure in O_3- and $PM_{2.5}$-exposed rats was enhanced and prolonged in rats with HFrD-induced metabolic syndrome.

Table S2 (Continued)

Type of study	Reference	Study period	Exposure dose/concentration	Study subject	Gender/Age	Major health outcomes
Epidemiological	Chen et al., 2013 [11]	1996-2010	Annual estimates of $PM_{2.5}$ exposure by assigning the 6-year mean concentration of $PM_{2.5}$ at each annual postal code	Human	35 years or older	Long-term exposure to $PM_{2.5}$ may contribute to the development of diabetes.
Epidemiological	Dijkema et al., 2011 [12]	2007	Traffic-related PM and NO_2	Human	50-75 years	No consistent associations between type 2 diabetes prevalence and exposure to TRAP was found, though there were some indications for a relation with traffic in a 250 m buffer.
Epidemiological	Ruckerl et al., 2014 [13]	2007-2008	NO, NO_2, CO, BC, $PM_{2.5}$, $PM_{2.5\text{-}10}$, and PM_{10}	Human	Slightly more males, with a mean age of approximately 62 years	Subjects with certain genes involved in the detoxification process render these people more susceptible to effects of PM mass on inflammation. Pre-diabetics were slightly more responsive to air pollution than diabetics, which might be explained by anti-diabetic medication that attenuates the effects of ambient air pollution on blood markers.
Epidemiological	Hou et al.,2016 [14]	N/A	Detected PAHs in the urine sample	Human	N/A	Urinary OH-PAHs levels were positively associated with type 2 diabetes among participants with impaired lung function. Higher levels of urinary OH-PAHs and reduced lung function had an additive effect on diabetes
Epidemiological	Ponticiello et al., 2015 [15]	N/A	Metal and PAHs	Human	Male traffic police: 48.2 years, female traffic policemen:45.6 years, male indoor workers: 48.2 years, female indoor workers: 44.9 years	Outdoor workers may be subject to an additional risk of developing obesity as a result of exposure to urban air pollution.
Epidemiological	Hu et al., 2015 [16]	2001-2008	PAHs	Human	18 years or older	Environmental exposure to PAHs independent of cigarette smoking is associated with insulin resistance, β-cell dysfunction, and increased prevalence of metabolic syndrome.
Epidemiological	Yang et al., 2017 [17]	2006–2008	Ambient PM_{10}, SO_2, NO_2 and O_3 from monitoring stations	Human	Adults/18–74 years	There were significant associations between air pollutants (PM_{10}, SO_2, NO_2, and O_3) and prehypertension and increased arterial BPs in adults. The associations were found to be stronger for females and the elderly.
Epidemiological	Chung et al., 2015 [18]	2009-2011	Particle number concentration: 17,000 number/cm^3, $PM_{2.5}$: 7.30 $\mu g/m^3$, BC: 0.68 $\mu g/m^3$ within 7 km from a major highway	Human	Male and female/average 58.5 years	Particle number concentration levels are associated with increased blood pressure.
Epidemiological	Thiering et al., 2013 [19]	N/A	NO_2:21.2-24.0 $\mu g/m^3$, PM_{10}:20.3-25.5 $\mu g/m^3$, $PM_{2.5}$: 13.3-17.5 $\mu g/m^3$ from traffic-related air pollution	Human	Children/10 years	Long term exposure of traffic-related air pollution may increase the risk of insulin resistance in children.
Epidemiological	Brook et al., 2013 [20]	2009-2010	$PM_{2.5}$ from urban: 11.5 ± 4.8 $\mu g/m^3$	Human	both male and female/18–50 years	$PM_{2.5}$, even at low levels, may reduce metabolic insulin sensitivity.
Controlled human exposure	Cosselman el al.,2012 [21]	N/A	Fine PM from diesel exhaust: 200 $\mu g/m^3$	Human	Male and female/18-49 years	Diesel exhaust inhalation was associated with a rapid, measurable increase in systolic but not diastolic BP in young nonsmokers, independent of perception of exposure.

Table S2 (Continued)

Type of study	Reference	Study period	Exposure dose/concentration	Study subject	Gender/Age	Major health outcomes
Toxicological	Matsuda et al., 2013 [22]	N/A	Concentrated $PM_{2.5}$: 600 μg/m^3	C57BL6 male mice	Male	No difference in body weight was observed across groups exposed to RC or HF diet and FA or EXP air exposure. Obese mice exposed to $PM_{2.5}$ (EXP-HF) had significantly higher fasting glycemia levels than FA-FH group (the group exposed to filtered air) The gene expression profile of adhesion molecules (E-selectin, VCAM-1, ICAM-1, cytokine IL-6, and MMP-9 (extracellular matrix metalloproteinase) was differently affected by $PM_{2.5}$.
Toxicological	Brocato et al., 2014 [23]	N/A	PM_{10}: 100μg/m^3, heavily concentrated with S, Al, Fe, Ni, Va, As, Pb, Cd, Mn, Ti, and Mg	FVB/N mice	Male/11 weeks	PM_{10}-induced genes involved in inflammation, cholesterol, lipid metabolism, and atherosclerosis.
Toxicological	Xu et al., 2016 [24]	N/A	$PM_{2.5}$ from road: 101.5±2.3 μg/m^3	C57BL/6J mice	Male/6-8 weeks	$PM_{2.5}$ inhalation results in the development of metabolic disorders and systemic inflammation through the NF-κB and Nrf2 signaling
Toxicological	Wang et al., 2017 [25]	N/A	Concentrated ambient $PM_{2.5}$ from University of Maryland, Baltimore area: 17-24 μg/m^3	Drosophila	Male and female	Drosophila developed systemic inflammation-like responses and dysregulated insulin signaling and have a markedly increased premature mortality in response to exposure to CAP.

[1.] N/A: information not available

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