Online Supplement

A comprehensive review of environmental risk factors and cardiovascular diseases

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Table S1. Epidemiological/observational evidence for an association between traffic noise and cardiovascular disease, events, and mortality with focus on recent studies.

| First author / year | Population / cohort | Noise sources | Major outcomes | Ref |
|---------------------|---------------------|---------------|----------------|-----|
| Roca-Barceló, 2021  | 21,936 CVD deaths   | Aircraft noise| CVD and CHD mortality risk tended to increase with increasing levels of aircraft noise ($L_{dn}$), while no linear trend was found for stroke mortality. | 1   |
| Kupcikova, 2021     | 502,651 subjects    | Road traffic noise | Road traffic noise exposure ($L_{den} > 65$ vs. $\leq 55$ dB(A)) led to $0.77\%$ (95% CI 0.60-0.95) higher SBP, $0.49\%$ (95% CI 0.32-0.65) higher DBP, $0.79\%$ (95% CI 0.11-1.47) higher triglycerides, and $0.12\%$ (95% CI −0.04-0.28) higher glycated hemoglobin. | 2   |
| Yankoty, 2021       | 1,065,414 subjects | Total environmental / transportation noise | The HRs for incident MI were 1.12 (95% CI 1.08-1.15), 1.11 (95% CI 1.07-1.14), and 1.10 (95% CI 1.06-1.14) per 10 $dB(A)$ increase in $L_{Aeq24}$, $L_{den}$, and $L_{night}$, respectively. | 3   |
| Gilani, 2021        | 909 subjects        | Road traffic noise | An OR of 2.25 (95% CI 1.38-3.67) for the prevalence of CAD per 5 $dB(A)$ increase in road traffic noise ($L_{den}$) was found. | 4   |
| Saucy, 2021         | 24,886 CVD deaths   | Aircraft noise | Acute increases in aircraft noise 2 hours preceding death were associated with total CVD mortality (OR 1.44, 95% CI 1.03-2.04) for the highest group of exposure ($L_{Aeq} > 50$ vs. $< 20$ dB). | 5   |
| Baudin, 2021        | 5,860 subjects      | Aircraft noise | Aircraft noise levels per 10 $dB(A)$ increase in $L_{night}$ increased the odds of antihypertensive medication by 43% (OR 1.43, 95% CI 1.19-1.73). | 6   |
| Osborne, 2020       | 498 subjects        | Combination of road traffic and aircraft noise | Higher noise exposure per 5 $dB(A)$ increase in $L_{Aeq24}$ predicted major CV events (HR 1.341, 95% CI 1.147-1.567). | 7   |
| Bai, 2020           | 37,441 cases of incident acute MI and 95,138 | Road traffic noise | Road traffic noise ($L_{Aeq24}$) per IQR increase was associated with an elevated risk of incident acute MI (HR 1.07, 95% CI 1.06-1.08) and CHF (HR, 1.07 95% CI 1.06-1.09). | 8   |
| Study                  | Subjects                          | Noise Source                | Findings                                                                                           |
|-----------------------|-----------------------------------|----------------------------|---------------------------------------------------------------------------------------------------|
| Thacher, 2020         | 52,758 subjects                  | Road traffic noise         | At the most exposed façade, road traffic noise per IQR increase was associated with a 13% (HR 1.13, 95% CI 1.06-1.19) and 11% (HR 1.11, 95% CI 0.99-1.25) higher CVD and stroke mortality, respectively. At the least exposed façade, road traffic noise remained to be associated with CVD (HR 1.09, 95% CI 1.03-1.15), IHD (HR 1.10, 95% CI 1.01-1.21), and stroke (HR 1.06, 95% CI 0.95-1.19) mortality. |
| Thacher, 2020         | 52,053 subjects                  | Road traffic noise         | There was no association between road traffic noise and filled prescriptions for antihypertensive drugs. |
| Andersson, 2020       | 6,304 men                        | Road traffic noise         | The HRs were 1.08 (95% CI 0.90-1.28) for CV mortality, 1.14 (95% CI 0.96-1.36) for IHD incidence, and 1.07 (95% CI 0.85-1.36) for stroke incidence in response to road traffic noise (L_{Aeq24} >60 vs. <50 dB). |
| Shin, 2020            | Subjects without a history of hypertension (701,174) or diabetes mellitus (914,607) | Road traffic noise         | An increase in L_{Aeq24} per 10 dB(A) was associated with an 8% increase in incident diabetes mellitus (HR 1.08, 95% CI 1.07-1.10) and a 2% increase in incident hypertension (HR 1.02, 95% CI 1.01-1.10). Similar estimates were obtained for L_{night}. |
| Baudin, 2020          | 6,105 subjects                   | Aircraft noise             | An increase per 10 dB(A) in L_{night} was associated with an increased risk of hypertension (RR 1.03, 95% CI 1.01-1.06). An association was also found between aircraft noise annoyance and hypertension risk (RR 1.06, 95% CI 1.00-1.13 for highly annoyed vs. not highly annoyed). |
| Pyko, 2019            | 20,012 subjects                  | Road traffic, railway, aircraft noise | In subjects exposed to all three traffic noise sources at ≥45 dB L_{den}, risks of IHD were elevated with a HR of 1.57 (95% CI 1.06-2.32), and a comparable observation for stroke (HR 1.42, 95% CI 0.87-2.32). |
| Héritier, 2019        | 4.4 million subjects             | Road traffic, railway, aircraft | MI mortality was increased in response to road traffic (HR 1.034, 95% CI 1.014-1.055), railway (HR 1.020, 95% CI 1.000-1.040). |
| Study | Year | Subjects/Subjects | Noise Type | Noise Description | Findings |
|-------|------|------------------|------------|-----------------|----------|
| Héritier, 2018 | 4.41 million subjects | Combination of road traffic, railway, aircraft noise | For the core night, the highest HR was observed for IHD mortality (1.025, 95% CI 1.016-1.034), while this association was lower for the daytime (1.018, 95% CI 1.009-1.028). HF mortality and daytime noise was associated with the highest HR (1.047, 95% CI 1.027-1.068). |
| Pyko, 2018 | 4,854 subjects | Road traffic, railway, aircraft noise | Aircraft noise increased the incident risk of hypertension by 16% (HR 1.16, 95% CI 1.08-1.24) per 10 dB increase in L_{den}. Road traffic and railway noise were not associated with incidence of hypertension. |
| Yang, 2018 | 663 subjects | Road traffic noise | Road traffic noise per 5 dB(A) increase was associated with the prevalence of CVD (OR 2.23, 95% CI 1.26-3.93). |
| Cai, 2018 | 21,081 incident CVD cases | Road traffic noise | No associations were found between road traffic noise and incident CVD, IHD, or CBVD in the total population. |
| Hahad, 2018 | 14,639 subjects | Road traffic, railway, aircraft noise | Traffic-related noise annoyance is associated with increased prevalence of AF. |
| Héritier, 2017 | 4.41 million subjects | Road traffic, railway, aircraft noise | HRs for MI mortality were per 10 dB increase in L_{den} 1.038 (95% CI 1.019-1.058) for road traffic, 1.018 (95% CI 1.004-1.031) for railway, and 1.026 (95% CI 1.004-1.048) for aircraft noise. |
| Zeeb, 2017 | 137,577 cases and 355,591 controls | Road traffic, railway, aircraft noise | There was no association between any of the traffic noise sources and incident hypertension. Likewise, no association between nighttime noise levels and hypertension was found. For the group of subjects with newly diagnosed hypertension followed by hypertensive heart disease, the ORs were elevated. |
| Fuks, 2017 | 41,072 subjects | Road traffic noise | A weak relationship between road traffic noise and incident self-reported hypertension was found, whereas no conclusive association with measured hypertension was established. |
| Study                      | Subjects/Populations                                    | Exposure/Noise Type               | Findings                                                                 |
|----------------------------|--------------------------------------------------------|-----------------------------------|--------------------------------------------------------------------------|
| Pitchika, 2017             | 2,552 subjects                                        | Road traffic noise                | No association between road traffic noise ($L_{Aeq24}$) and prevalent hypertension was found. |
| Roswall, 2017              | 50,744 subjects                                       | Road traffic noise                | Road traffic noise was associated with a higher risk of MI, with a HR of 1.14 (95% CI 1.07-1.21) per IQR increase in $L_{den}$. |
| Evrard, 2017               | 1,244 subjects                                        | Aircraft noise                    | Only in men, a 10 dB(A) increase in aircraft noise ($L_{night}$) was associated with risk of hypertension (OR of 1.34, 95% CI 1.00-1.97). |
| Dimakopoulou, 2017         | 780 subjects                                           | Aircraft noise                    | A 10 dB increase in $L_{night}$ resulted in an OR of 2.63 (95% CI 1.21-5.71) for hypertension and of 2.09 (95% CI 1.07-4.08) for doctor-diagnosed cardiac arrhythmia. |
| Sørensen, 2017             | 57,053 subjects                                       | Road traffic noise                | An IRR of 1.14 for HF (95% CI 1.08-1.21) per IQR increase in $L_{den}$ road traffic noise was found. |
| Seidler, 2016              | 19,632 cases and 834,734 controls                     | Road traffic, railway, aircraft noise | A 10 dB increase in $L_{Aeq24}$ was associated with higher odds of MI in response to road traffic (2.8%, 95% 1.2-4.5) and railway noise (2.3%, 95% CI 0.5-4.2), but not aircraft noise. Aircraft noise levels of 60 dB and above were associated with increased MI risk (OR 1.42, 95% CI 0.62-3.25). |
| Recio, 2016                | Cohort of subjects ≥65 years                           | Road traffic noise                | Short-term road traffic noise increased the risk of death from IHD, MI, and CBVD. |
| Monrad, 2016               | 57,053 subjects                                       | Road traffic, railway noise       | A 10 dB increase in $L_{den}$ road traffic noise was associated with a 6% increased risk of AF (IRR 1.06, 95% CI 1.00-1.12), which was weaker after further adjustment for air pollutants. AF risk was not related to railway noise. |
| Sørensen, 2011             | 57,053 subjects                                       | Road traffic noise                | An IRR of 1.14 for stroke (95% CI 1.03-1.25) per 10 dB increase in $L_{den}$ road traffic noise was found. |
| Beelen, 2009               | 120,852 subjects                                      | Road traffic noise, traffic intensity | Traffic intensity was associated with CV mortality, with highest RR of 1.11 (95% CI 1.03-1.20 per increase in 10,000 motor vehicles/24 h). Road traffic noise (>65 dB(A)) was associated with increased risk of IHD (RR 1.15, 95% CI 0.86-1.53) and HF mortality (RR 1.99, 95% CI 1.05-3.79), |
which was attenuated after further adjustment air pollution and traffic intensity.

CVD: Cardiovascular disease, CHD: Coronary heart disease, $L_{dn}$: Day-night noise levels, SBP: Systolic blood pressure, DPB: Diastolic blood pressure, HR: Hazard ratio, MI: Myocardial Infarction, $L_{Aeq(time \ period)}$: Noise levels over a certain period of time, $L_{night}$: Night noise levels, IHD: Ischemic heart disease, CHF: Congestive heart failure, IQR: Interquartile range, CBVD: Cerebrovascular disease, dB: Decibel, OR: Odds ratio, CI: Confidence interval, CAD: Coronary artery disease, $L_{den}$: Day-evening-night noise levels, AF: Atrial fibrillation, IRR: Incidence rate ratio, RR: Relative risk
Table S2. Human studies on the association of atherosclerosis, vascular (endothelial) dysfunction, inflammation, or oxidative stress with ambient air pollution or traffic noise with focus on recent studies.

| First author / year | Population / cohort | Air pollutants | Major outcomes | Ref |
|---------------------|---------------------|----------------|----------------|-----|
| Riggs, 2021         | 73 subjects with moderate to high CVD risk | PM$_{2.5}$, O$_3$ | An IQR increase in PM$_{2.5}$ was associated with augmentation pressure (3.1 mmHg), pulse pressure (5.9 mmHg), and aortic systolic pressure (8.1 mmHg). An IQR increase in O$_3$ was positively associated with augmentation index (5.5%), augmentation pressure (3.1 mmHg), and aortic systolic pressure (10 mmHg). | 34 |
| Liu, 2021           | 40 chronic obstructive pulmonary disease patients and 75 controls | PAHs | A 1-fold increase in hydroxylated PAHs was associated with a 4.1-15.1% elevation of malonaldehyde, which was stronger in subjects with impaired lung function. | 35 |
| Ni, 2021            | 740 subjects | PM$_{2.5}$ | Acute increases in PM$_{2.5}$ were associated with increased soluble lectin-like oxidized LDL receptor-1, but not with nitrite. | 36 |
| Nassan, 2021        | 456 men | PM$_{2.5}$ species | Acute increases in PM2.5 species were associated with metabolic pathways involved in inflammation, oxidative stress, immunity, and nucleic acid damage and repair. | 37 |
| Mann, 2021          | 299 children | traffic-related air pollutants (sum of PAH456, NO$_2$, elemental carbon, PM$_{2.5}$) | Acute increases in traffic-related air pollutants were associated with 8-isoprostane. | 38 |
| Hennig, 2020        | 4,814 subjects | PM$_{2.5}$, PM$_{10}$, NO$_2$ | Air pollutants were marginally associated with progression of atherosclerosis (carotid intima media thickness, coronary calcification, thoracic aortic calcification) in subjects with no or low baseline atherosclerotic burden. | 39 |
| Hennig, 2020        | 4,814 subjects | Particle number | Per IQR increase in particle number and road traffic noise | 40 |
| Study       | Subjects | Pollutants | Findings                                                                                          |
|------------|----------|------------|--------------------------------------------------------------------------------------------------|
| Prunicki, 2020 | 100 subjects | PM$_{2.5}$, NO, NO$_2$, CO, PAHs | Air pollutants were associated with acute inflammation, oxidative stress, endothelial dysfunction, altered hemostasis, diastolic blood pressure, and monocyte enrichment. |
| Salimi, 2020  | 615 subjects | PM$_{2.5}$, PM$_{10}$ | PM$_{2.5}$ ($-0.09$, 95% CI $-0.15$ - $-0.03$) and PM$_{2.5}$ ($-0.07$, 95% CI $-0.13$ - $-0.09$) were inversely associated with brachial artery flow-mediated dilation. |
| Riggs, 2020    | 100 subjects | PM$_{2.5}$ | A 10 μg/m$^3$ increase in PM$_{2.5}$ was associated with a 12.4% decrease in reactive hyperemia index (95% CI $-21.0$ - $-2.7$). Increased PM$_{2.5}$ was associated with elevated F-2 isoprostane metabolite, angiopoietin 1, vascular endothelial growth factor, placental growth factor, intracellular adhesion molecule-1, and matrix metalloproteinase-9 as well as reduced vascular adhesion molecule-1. |
| Li, 2019       | 73 subjects | PM$_{2.5}$, BC, NO$_2$, CO | Increases in air pollutants were associated with reductions in circulating high-density lipoprotein cholesterol and apolipoprotein A-I as well as elevations in HDL oxidation index, oxidized LDL, malondialdehyde, and C-reactive protein. |
| Lin, 2019      | 26 subjects | PAHs      | Increases in 5-, 12-, and 15-hydroxyeicosatetraenoic acid as well as 9- and 13-hydroxyoctadecadienoic acid were observed. Decreases in paraoxonase and arylesterase as well increases in C-reactive protein and fibrinogen were observed. |
| Yang, 2019     | 364 subjects | PM$_{2.5}$ | Increases in PM$_{2.5}$ were related to higher incidence of high-risk plaque (HR 1.62, 95% CI 1.22-2.15), formation of either fibrofatty or necrotic core component in newly developed |
| Study | Participants | Exposure | Findings |
|-------|--------------|----------|----------|
| Morishita, 2019 | 50 subjects | Particle number, BC | Acute increases in particle number and BC were associated with increases in aortic augmentation pressure and trend toward lower reactive hyperemia index. |
| Balmes, 2019 | 87 subjects | O$_3$ | Acute O$_3$ exposure did not alter C-reactive protein, monocyte-platelet conjugates, and microparticle-associated tissue factor activity, whereas increases in endothelin-1 and decreases in nitrotyrosine were observed. |
| Han, 2019 | 60 subjects with prediabetes and 60 healthy subjects | PM$_{2.5}$ | Acute exposure to PM$_{2.5}$ resulted in increased exhaled nitric oxide, white blood cells, neutrophils, interleukin-1α, and glycated hemoglobin. Compared to healthy subjects, pre-diabetic subjects displayed pronounced PM$_{2.5}$-associated systemic inflammation, elevated systolic and diastolic blood pressure, impaired endothelial function, and elevated fasting glucose. |
| Xia, 2019 | 215 pregnant women | PM$_{2.5}$ | Acute increases in PM$_{2.5}$ and lead constituent was associated with endothelial dysfunction (increased endothelin-1, E-selectin, and intracellular adhesion molecule-1) and inflammation (increased interleukin-1β, interleukin-6, tumor necrosis factor-α). Elevated inflammation and endothelial dysfunction were partially mediated by the effect of PM$_{2.5}$ and lead constituent on blood pressure. |
| Li, 2019 | 3,820 subjects | PM$_{2.5}$, BC, O$_3$, sulfate, NO$_X$ | Negative associations of acute PM$_{2.5}$ and BC with P-selectin, of O$_3$ with monocyte chemoattractant protein 1, and of sulfate and NO$_X$ with osteoprotegerin were found. |
| Ljungman, 2018 | 5,842 subjects | Proximity to roadway, PM$_{2.5}$, particle number, sulfate, O$_3$ | Living in the vicinity of a major roadway was associated with higher arterial stiffness (carotid-femoral pulse-wave velocity). No associations were found for PM$_{2.5}$, particle number, sulfate, or O$_3$. |
| Zhang, 2018 | 4,544 subjects | PM$_{2.5}$, PM$_{10}$ | PM$_{2.5}$ and PM$_{10}$ were associated with a higher prevalence of |
| Study                  | Sample Size | Exposure                | Description                                                                                                                                                                                                 | Reference |
|-----------------------|-------------|-------------------------|------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------|-----------|
| Choi, 2018            | 6,430 patients who underwent intracoronary acetylcholine provocation test | PM\textsubscript{10} | PM\textsubscript{10} was associated with coronary artery spasm and transient ST-segment elevation during the acetylcholine provocation test.                                                               | 54        |
| Dorans, 2017          | 3,506 subjects | PM\textsubscript{2.5} | The presence/extent of thoracic aortic calcium Agatston score or abdominal aortic calcium Agatston score was not consistently associated with PM\textsubscript{2.5} or major roadway proximity.                  | 55        |
| Li, 2017              | 3,996 subjects | PM\textsubscript{2.5}, sulfate, NO\textsubscript{x}, BC, O\textsubscript{3} | Acute increases in PM\textsubscript{2.5} and sulfate were associated with increased C-reactive protein, which was also true for NO\textsubscript{x} in case of interleukin-6 and for BC, sulfate, and O\textsubscript{3} in case of tumor necrosis factor receptor 2. Conversely, BC, sulfate, and NO\textsubscript{x} were negatively associated with fibrinogen, and sulfate was negatively associated with tumor necrosis factor \( \alpha \). | 56        |
| Day, 2017             | 89 subjects | O\textsubscript{3} | Acute increases in O\textsubscript{3} per 10 ppb increase were associated with increased soluble P-selectin (36.3%, 95% CI 29.9-43.0), diastolic blood pressure (2.8%, 95% CI 0.6-5.1), exhaled nitric oxide (18.1%, 95% CI 4.5-33.5), exhaled breath condensate nitrite and nitrate (31.0%, 95% CI 0.2-71.1), and decreased augmentation index (−9.5%, 95% CI −17.7 - −1.4). | 57        |
| Mirowsky, 2017        | 13 subjects with CAD | O\textsubscript{3} | Per acute IQR increase in O\textsubscript{3}, changes were determined for plasminogen activator inhibitor-1 (40.5%, 95% CI 8.7-81.6), tissue plasminogen factor (6.6%, 95% CI 0.4-13.2), monocytes (10.2%, 95% CI 1.0-20.1), interleukin-6 (15.9%, 95% CI 3.6-29.6), neutrophils (8.7%, 95% CI 1.5-16.4), baseline diameter of the brachial artery (−2.5%, 95% CI −5.0- | 58        |
The probability of having increased brachial-ankle pulse wave velocity was higher in inactive subjects with higher PM$_{10}$, PM$_{2.5}$, NO$_2$, particle number, and lung deposited surface area.

PM$_{2.5}$ and NO$_X$ were associated with progression of coronary calcification.

Episodic increases in PM$_{2.5}$ were related to higher endothelial cell apoptosis, elevated circulating monocytes, increased T (but not B) lymphocytes, and an anti-angiogenic plasma profile.

Reactive hyperemia index was inversely associated with acute increases PM$_{2.5}$, BC, NO$_X$, and CO with high oxidative potential.

PM$_{2.5}$ from dust/soil and several crustal and transition metals (including magnesium, iron, strontium, cobalt, titanium) were associated with increases in endothelin-1. Manganese, potassium, and CO were associated with increases in intracellular adhesion molecule-1. PM$_{2.5}$ from industry and metal cadmium was associated with decreased vascular cell adhesion molecule 1.

Acute increases in PM$_{2.5}$ were associated with brachial-ankle pulse wave velocity, whereas no association was found for NO$_2$. NO$_2$ was associated with increased C-reactive protein.

Acute increases in BC were associated with increased arterial stiffness.

| First author / year | Population / cohort | Noise sources | Major outcomes | Ref |
|---------------------|---------------------|---------------|----------------|-----|
| Schmidt, 2021       | 70 subjects with CVD | Aircraft noise | Acute aircraft noise exposure at night impaired endothelial function (flow-mediated dilation) and cardiac diastolic | 66 |

### Traffic noise

- **Schmidt, 2021**: 70 subjects with CVD, Aircraft noise
  - Acute aircraft noise exposure at night impaired endothelial function (flow-mediated dilation) and cardiac diastolic
| Study             | Sample Size | Noise Source                                           | Findings                                                                                                                                                                                                 |
|-------------------|-------------|--------------------------------------------------------|---------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------|
| Hahad, 2021       | 5,000 subjects | Aircraft, railway noise                                | Aircraft and railway noise annoyance were associated with increased midregional pro atrial natriuretic peptide, which predicted incident CVD.                                                               |
| Biel, 2020        | 46 subjects  | Total environmental noise (traffic noise included)     | Acute increases in both air pollution and noise were associated with endothelial function and heart rate variability.                                                                                  |
| Eze, 2020         | 1,389 subjects | Road traffic, railway, aircraft noise                  | Both air pollution and traffic noise were associated with DNA methylation, with both distinct and shared enrichments for pathways linked to cellular development, immune responses, and inflammation. |
| Thiesse, 2020     | 26 subjects  | Road traffic noise                                     | After sleeping with highly intermittent road traffic noise, evening cortisol levels were elevated.                                                                                                       |
| Herzog, 2019      | 70 subjects  | Railway noise                                          | Acute railway noise exposure was associated with impaired flow-mediated dilation. Proteomic analysis indicated substantial changes of plasma proteins in response to noise centered on proinflammatory, redox, and pro-thrombotic pathways. |
| Cai, 2017         | 144,082 subjects | Road traffic noise                                    | An IQR increase in L_{day} road traffic noise was associated with 0.7% (95% CI 0.3-1.1) higher triglycerides, 1.1% (95% CI 0.02-2.2) higher C-reactive protein, and 0.5% (95% CI 0.3-0.7) higher high-density lipoprotein, with only the latter being robust to further control for air pollution. |
| Foraster, 2017     | 2,775 subjects | Road traffic, railway, aircraft noise                  | A 0.87% (95% CI 0.31-1.43) increase in brachial-ankle pulse wave velocity per IQR increase in L_{den} railway noise was observed. Total number of noise events at night, but not at day, was related to brachial-ankle pulse wave velocity. |
| Lefèvre, 2017     | 1,244        | Aircraft noise                                         | Aircraft noise was associated with cortisol in the evening.                                                                                                                                               |
| Halonen, 2017     | 2,592 subjects | Road traffic noise                                     | A 9.1μm (95% CI –7.1-25.2) increase in carotid intima-media thickness per 10 dB(A) increase in L_{night} was observed.                                                                                   |
| Author(s), Year | Subjects | Noise Exposure | Effects | Reference |
|----------------|----------|----------------|---------|-----------|
| Schmidt, 2015  | 60 subjects at increased risk of CVD | Aircraft noise | Acute aircraft noise exposure at night impaired endothelial function and increased systolic blood pressure. | 76 |
| Sørensen, 2015 | 39,863 subjects | Road traffic noise | Slightly higher cholesterol may be linked to road traffic noise. | 77 |
| Schmidt, 2013  | 75 subjects | Aircraft noise | Acute aircraft noise exposure at night was associated with impaired flow-mediated dilation, which was attenuated by the administration of Vitamin C. Adrenaline was increased and pulse transit time decreased in response to noise. | 78 |

CVD: Cardiovascular disease, PM\(_{(\text{diameter size})}\): Particulate matter, \(\text{NO}_2\): Nitrogen dioxide, IQR: Interquartile range, \(L_{\text{night}}\): Night noise levels, OR: Odds ratio, CI: Confidence interval, \(O_3\): Ozone, \(\text{NO}_x\): Nitrogen oxides, HR: Hazard ratio, BC: Black carbon, CO: Carbon monoxide, PAHs: Polycyclic aromatic hydrocarbons, NO: Nitrogen monoxide, CAD: Coronary artery disease, \(L_{\text{den}}\): Day-evening-night noise levels, \(L_{\text{day}}\): Day noise levels
Table S3. Animal in vivo studies on non-auditory noise effects on cardiovascular and endothelial dysfunction, inflammation, or oxidative stress. Only articles that are not mentioned in the main article text and used <100 dB average sound pressure level are listed here.

| First author / year | Animals and model | Noise scenario | Major outcomes | Ref |
|---------------------|-------------------|----------------|----------------|-----|
| Borg, 1981          | Sprague-Dawley, spontaneously hypertensive rats (SHR) | 80 - 100 dB (noise type unknown), 10 h/d for entire lifespan | Noise exposure caused a shorter lifespan and higher frequency of CVD in spontaneously hypertensive rats as compared to normotensive rats. | 79 |
| Peterson, 1984      | Rhesus monkeys    | 85 dB (realistic noise sequence), 24 h/d for 6 months | Noise exposure caused a substantial increase in blood pressure as well as disruption of the diurnal rhythm of heart rate, blood pressure, and caused "pauses" in cardiac rhythm. | 80 |
| Peterson, 1984      | Macaque monkeys   | 87 - 90 dB (construction noise), 4 - 8 h/d for 97 d | Noise exposure caused an increase in blood pressure by 8.2% (4 h/d scenario) and 16.5% (8 h/d scenario). Whereas blood pressure increases persisted after noise cessation, the heart rate returned to baseline. | 81 |
| Kirby, 1984         | Macaque monkeys   | 95 dB (broadband noise), 30 min | Noise exposure caused a more pronounced increase in blood pressure in the offspring of hypertensive monkeys, whereas heart rate was significantly changed. Also the resting blood pressure in the offspring of hypertensive monkeys was higher than offspring of normotensive monkeys. | 82 |
| Wu, 1992            | Rats              | 85 - 95 dB (unknown noise type), 12 - 16 h/d for 4 - 8 weeks | Noise exposure impaired endothelium-dependent vasodilation as determined by acetylcholine (ACh)-response in the isolated thoracic aorta. Noise also increased the sensitivity to the vasoconstrictor serotonin, but not phenylephrine or potassium chloride, and increased systolic blood pressure by 31 mmHg. | 83 |
| Altura, 1992        | Rats              | Up to 100 dB (broadband noise), 4 h/d for 2 - 4 weeks | Noise exposure led to increased systolic and diastolic blood pressure (16 mmHg) along with magnesium deficiency and reduced lumen sizes of microvessels. | 84 |
| Morvai, 1989        | CFY rats          | 95 dB (industrial) | Noise exposure lowered cardiac output and hepatic blood flow. | 85 |
| Year       | Species                  | Noise Duration/Condition                          | Summary                                                                                     |
|------------|--------------------------|--------------------------------------------------|-----------------------------------------------------------------------------------------------|
| 1994       | Rats                     | Up to 100 dB (broadband noise), 4 h/d for 3 - 4 weeks | Noise exposure increased systolic blood pressure by 25 mmHg (3 weeks noise) and by 37 mmHg (4 weeks noise), which was associated with pronounced endothelial dysfunction in isolated mesenteric arterial rings. |
| 1994       | Wistar rats and SHR      | 65 dBA (low frequency noise, 4 and 250 Hz), 24 h/d for 52 weeks | Noise exposure was associated with significantly increased microvessel wall area, number of microvessels with an outer diameter > 19 microns, the degree of cardiac fibrosis, and the extent of ischemic myocardial lesions in SHR, but not in normotensive rats. Noise did not alter cardiac weights and dimensions, heart rate, and dp/dtmax. |
| 2000       | Wistar-Kyoto rats and SHR| 95 dB (noise type unknown), 3 min                | Noise exposure led to a tetrodotoxin-sensitive increase in glutamate release in the amygdala of SHR, but not normotensive rats. Also pressor response to noise was enhanced in SHR, all of which indicates an exaggerated stress response of glutamatergic neurons in the amygdala of SHR as compared to normotensive rats. |
| 2007       | Rats                     | 90 dB (noise type unknown), 15 min/d for 3 – 5 weeks | Noise exposure impaired the microvascular integrity (mesenteric arteries) in rats as revealed by significantly more leaks per venule length and greater leak area per venule length. Co-treatment with vitamin E plus a-lipoic acid or Traumeel (a homeopathic anti-inflamatory-analgesic) partly prevented these adverse effects of noise. |
| 2013       | Rats                     | 90 dB (low frequency noise, ≤ 500 Hz) for 3 months | Noise exposure caused significant myocardial fibrosis (increased collagen deposition between the cardiomyocytes) in rats. Also connexin43/muscle ratio was decreased by noise. Transmission electron microscopy also revealed noise-induced changes of cardiomyocyte ultrastructure, e.g. altered interstitial collagen deposits and changes in mitochondria and intercalated discs of the cardiomyocytes. |
| 2013       | Wistar rats              | 70 – 80 dB (octave-band noise (8-16 | Noise exposure increased corticosterone levels, affected various parameters of the endocrine glands and cardiac function. Markers of |
| **Ersoy, 2014** | Albino rats | Noise type and protocol unknown | Noise exposure significantly decreased superoxide dismutase expression in the cerebral cortex but increased malondialdehyde levels in the brainstem and cerebellum. Rosuvastatin increased superoxide dismutase expression in the cerebral cortex and brain stem, but significantly decreased malondialdehyde values in the brain stem. | 94 |
| **Gannouni, 2014** | Wistar rats | 70 dB (noise type unknown) | Noise exposure caused time-dependent changes in the morphological structure of the adrenal cortex involving disarrangement of cells and modification in thickness of the different layers of the adrenal gland. These observations are compatible with noise-induced changes of the morphological structure of heart tissue causing irreversible cell damage and leading to necrosis or cell death. | 95 |
| **Said, 2016** | Albino rats | 80 - 100 dB (chronic and intermittent octave band noise, 8-16 kHz), 8 h/d for 20 d | Noise exposure adversely affected the cardiovascular system by increased levels of circulating stress hormones (e.g. corticosterone, adrenaline, noradrenaline, endothelin-1). Noise also negatively affected oxidative stress markers (e.g. higher malondialdehyde levels and decreased superoxide dismutase expression). These data are compatible with endothelial dysfunction, which was further supported by impaired nitric oxide metabolism and elevated blood pressure in noise-exposed rats. | 96 |
| **Cui, 2016** | Rats | Up to 100 dB (octave band noise, 0.4-6.3 kHz), 4 h/d for 30 d | Noise exposure caused a transient increase in markers of inflammation, blood glucose, triglycerides, and alterations in the microbiome that returned to baseline at 14 d after noise exposure cessation. | 97 |
| **Kvandova, 2020** | C57BL/6 mice and Ogg1-/- mice | 72 dB (aircraft noise), 24 h/d for 4 d | Noise exposure induced oxidative DNA damage that was associated with enhanced leucocyte oxidative burst activity and other markers of inflammation (e.g. cyclooxygenase-2 as well as oxidative stress | 98 |
(e.g. 4-hydroxynonenal, 3-nitrotyrosine levels and NOX-2 expression). Noise impaired endothelial function (ACh-response) but not endothelium-independent relaxation (nitroglycerin-response). Genetic deficiency in 8-oxoguanine glycosylase knockout (Ogg1<sup>-/-</sup>) further aggravated most of these adverse noise effects and induced a significant impairment of the endothelium-independent relaxation (nitroglycerin-response).

Table S4. Epidemiological/observational evidence for an association between ambient air pollution and cardiovascular disease, events, and mortality with focus on recent studies.
| First author / year | Population / cohort | Air pollutants | Major outcomes | Ref |
|---------------------|---------------------|----------------|----------------|-----|
| Slawsky, 2021       | 5,681 patients who underwent cardiac catheterization | PM$_{2.5}$ from ammonium bisulfate and ammonium nitrate | OR of CAD increased by 20% (95% CI 1.11-1.30) per IQR increase in PM$_{2.5}$ from ammonium bisulfate. Ammonium nitrate was associated with a 18% increased OR of CAD (95% CI 1.05-1.32) per IQR increase in PM$_{2.5}$ from ammonium nitrate. Results for MI were broadly similar to those of CAD. | 99 |
| Aturinde, 2021      | 538,573 hospital admissions | BC, CO, PM$_{10}$, PM$_{2.5}$, SO$_x$ | There were significant place-specific associations between air pollutants and CVD admissions. | 100 |
| Wang, 2021          | 7 cities | PM$_{10}$ | A 10 μg/m$^3$ acute increase in PM$_{10}$ was associated with a 0.13% (95% CI −0.01-0.26%) increase in CV emergency ambulance calls. | 101 |
| Cao, 2021           | 32,135 subjects | PM$_{2.5}$ | A positive relationship between PM$_{2.5}$ and stage 1 hypertension (SBP: 130-139 mmHg or DBP: 80-89 mmHg; OR 1.05, 95% CI 1.02-1.08 per 10 μg/m$^3$ increase) was observed. | 102 |
| Leili, 2021         | 2,091 hospital admissions | PM$_{2.5}$ | An acute increase in PM$_{2.5}$ was associated with increased risk of hospitalization due to HF and MI. | 103 |
| Shin, 2021          | 92,567 acute MI patients | PM$_{2.5}$ | An acute increase in PM$_{2.5}$ was associated with acute MI. | 104 |
| Liao, 2021          | 96,582 subjects with a history of stroke or acute MI | PM$_{2.5}$ | Analyses restricted to PM$_{2.5}$-levels <12 μg/m$^3$ showed increased risk of CVD mortality (HR 2.31, 95% CI 1.96-2.71), stroke (HR 1.41, 95% CI 1.09-1.83), and acute MI (HR 1.51, 95% CI 1.21-1.89) per 10 μg/m$^3$ increase in PM$_{2.5}$. | 105 |
| Dehom, 2021         | 93,857 renal transplant recipients | PM$_{2.5}$ | PM$_{2.5}$ was associated with increased risk of CVD (HR 2.38, 95% CI 1.94-2.92 per 10 μg/m$^3$ increase) and CHD mortality (HR 3.10, 95% CI 1.96-4.90 per 10 μg/m$^3$ increase). | 106 |
| Kaihara, 2021       | 835,405 acute CVD hospital | PM$_{2.5}$ | Acute increases in PM$_{2.5}$ was associated with increased CVD hospitalizations as well as higher hospitalization durations | 107 |
| Study                          | Population Details                                                                 | Outcomes Description                                                                                                                                                                                                 | Reference |
|-------------------------------|-------------------------------------------------------------------------------------|----------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------|-----------|
| Ren, 2021                     | 332,090 CVD admissions                                                              | A 10 μg/m³ acute increase in PM$_{2.5}$ was associated with increased CVD hospital admissions (1.23%, 95% CI 1.01-1.45).                                                                                                   | 108       |
| Du, 2021                      | 23,256 subjects                                                                    | IQR increases in PM$_{2.5}$, PM$_{10}$, and SO$_2$ were associated with SBP (changes: 0.64-1.86 mmHg), DBP (changes: 0.35-0.70 mmHg), and the prevalence of hypertension (ORs 1.09-1.19). | 109       |
| Xu, 2021                      | 5,143 post-CVD patients                                                             | Increased CVD (HR 1.57, 95% CI 1.27-1.94) and MI mortality (HR 1.82, 95% CI 1.16-2.83) in response to PM$_{2.5}$.                                                                                                           | 110       |
| Raza, 2021                    | 34,748 subjects                                                                    | Increased risk of IHD among subjects with increased PM$_{2.5}$ and PM$_{10}$ exposure.                                                                                                                              | 111       |
| Thabethe, 2021                | 3 cities                                                                            | Increased risk of CVD mortality in response to PM$_{10}$.                                                                                                                                                              | 112       |
| Rahman, 2021                  | 340,758 CVD emergency department visits, 253,407 hospital admissions, and 16,858 deaths | Increases in CVD emergency department visits (0.27%, 95% CI 0.07-0.47), hospitalizations (0.32%, 95% CI 0.08-0.55), and deaths (0.87%, 95% CI 0.27-1.47) per 10 μg/m³ increase in PM$_{2.5}$. | 113       |
| Raza, 2021                    | 2,221 subjects                                                                     | PM$_{2.5}$ levels above the median increased the risk of IHD by 13% (95% CI 17-45) and the risk of stroke recurrence by 21% (95% CI 19-80).                                                                                                    | 114       |
| Kim, 2021                     | 1,469,972 subjects                                                                 | Among physical active young adults exposed to high levels of PM$_{2.5}$ or PM$_{10}$, the risk of CVD was pronounced.                                                                                                 | 115       |
| Meng, 2021                    | 398 cities and 19.7 million CV deaths                                                | An acute increase in NO$_2$ per 10 μg/m³ increase was associated with a 0.37% (95% CI 0.22-0.51) increase in CV mortality.                                                                                             | 116       |
| Sepandi, 2021                 | 69,000 emergency hospital admissions for                                            | The cumulative RRs for emergency hospital admissions for CVD were 1.13 (95% CI 1.01-1.26), 1.15 (95% CI 1.02-1.29), and 1.08 (95% CI 1.01-1.18) for CO, NO$_2$, and PM$_{2.5}$, respectively. | 117       |
| Reference         | Sample Size                  | Exposure | Outcomes                                                                 |
|-------------------|------------------------------|----------|---------------------------------------------------------------------------|
| Khajavi, 2021     | 4,580 subjects               | PM$_{10}$ | PM$_{10}$ increased the risk of incident hypertension (HR 1.96, 95% CI 1.48-2.62). |
| Alexeeff, 2021    | 169,714 patients with COPD  | PM$_{2.5}$ | A 10 µg/m$^3$ increase in PM$_{2.5}$ was associated with an elevated risk of CV mortality (HR 1.10, 95% CI 1.01-1.20). |
| Coleman, 2021     | 5,591,168 cancer patients   | PM$_{2.5}$ | PM$_{2.5}$ increased CV mortality (HR 1.32, 95% CI 1.26-1.39). |
| Lin, 2021         | 28,548 outpatient clinic visits for hypertension | PM$_{2.5}$ | Acute increases in PM$_{2.5}$ was associated with increased outpatient clinic visits for hypertension. |
| Yazdi, 2021       | 63,006,793 subjects         | PM$_{2.5}$, NO$_2$, O$_3$ | PM$_{2.5}$ was associated with an increased risk of hospital admissions for MI, ischemic stroke, and AF with the highest effect seen for stroke. |
| Wang, 2021        | 432,530 subjects            | PM$_{2.5}$, PM$_{10}$, PM$_{2.5-10}$, NO$_2$, NO$_x$ | HRs of HF for a 10 µg/m$^3$ increase in PM$_{2.5}$, PM$_{10}$, PM$_{2.5-10}$, NO$_2$, and NO$_x$ were 1.85 (95% CI 1.34-2.55), 1.61 (95% CI 1.30-2.00), 1.13 (95% CI 0.80-1.59), 1.10 (95% CI 1.04-1.15), and 1.04 (95% CI 1.02-1.06), respectively. |
| Fasola, 2021      | 1,585 subjects              | PM$_{2.5}$, PM$_{10}$, NO$_2$ | Acute increases in PM$_{2.5}$, PM$_{10}$, and NO$_2$ were associated with increased risk of CV hospitalization. |
| Li, 2021          | 10,466 HF hospitalizations  | PM$_{2.5}$ | Acute increases in PM2.5 were associated with increased risk of HF hospitalization. |
| Sokoty, 2021      | 43,424 patients with CVD   | CO        | Acute increases in CO increased the incidence of CV hospitalization. |
| Abohashem, 2021   | 503 subjects                | PM$_{2.5}$ | PM$_{2.5}$ was associated with major adverse CV events (HR 1.404, 95% CI 1.135-1.737). |
| Pang, 2021        | 34,040 subjects             | Traffic-related air pollution | Higher odds of valvular heart disease in response to traffic-related air pollution. |
| Sui, 2021         | 27,431 CV                   | PM$_{2.5}$ | An acute increase per 10 µg/m$^3$ in PM$_{2.5}$ was related to a... |
| Study                | Data Points                                                                 | PM Metric | Description                                                                                           |
|---------------------|------------------------------------------------------------------------------|-----------|-------------------------------------------------------------------------------------------------------|
| deSouza, 2021       | 3,666,657 CV hospitalizations among Medicaid adults                           | PM$_{2.5}$ | A 0.9% (95% CI 0.6-1.1) increase in CV admission rates per 10 µg/m$^3$ acute increase in PM$_{2.5}$ was observed. |
| Xu, 2021            | 31,462 subjects                                                              | PM$_{2.5}$ | OR of hypertension was 1.08 (95% CI, 1.04-1.12) per 10 µg/m$^3$ increase in PM$_{2.5}$.               |
| Li, 2021            | Citywide study                                                               | O$_3$     | An acute increase in O$_3$ per 10 µg/m$^3$ was associated with a 0.59% (95% CI 0.30-0.88) higher risk CVD death. |
| Kim, 2020           | 196,167 subjects                                                             | PM$_{2.5}$ | A 10 µg/m$^3$ increase in PM$_{2.5}$ was associated with 4 and 10% increases in the incidence of total CVD (95% CI 0-9) and IHD (95% CI 4-16), respectively. |
| Zhang, 2020         | 178,780 subjects                                                             | PM$_{2.5}$, PM$_{10}$ | PM$_{2.5}$ and PM$_{10}$ were associated with increased incidence of arrhythmias.                        |
| Elliott, 2020       | 104,990 females                                                              | PM$_{2.5}$ | PM$_{2.5}$ was associated with increased risk of CVD (HR 1.09, 95% CI 0.99-1.20).                        |
| Stafoggia, 2020      | 2,154,810 CVD hospitalizations                                               | PM$_{2.5}$, PM$_{10}$ | Relative increases of total CVD admissions per 10 µg/m$^3$ acute variation in PM$_{10}$ and PM$_{2.5}$ were 0.55% (95% CI 0.32-0.77) and 0.97% (0.67-1.27), respectively. |
| Wu, 2020            | 26,749 CV events                                                             | PM$_{2.5}$ | Acute increases in PM$_{2.5}$ was associated with elevated risk of CV events.                          |
| Prabhakaran, 2020    | 5,342 subjects                                                               | PM$_{2.5}$ | Long-term exposures of 1, 1.5, and 2 years increased the risk of incident hypertension by 53% (HR 1.53, 95% CI 1.19-1.96), 59% (HR 1.59, 95% CI 1.31-1.92), and 16% (HR 1.16, 95% CI 0.95-1.43) per IQR increase in PM$_{2.5}$, respectively. |
| Quintyne, 2020       | Citywide CV hospitalizations                                                  | Air quality index for health | Air quality is related to CV hospital admissions.                                                      |
| Bi, 2020            | 1,172,516 CVD emergency department visits                                    | PM$_{2.5}$ | Temporal changes in the risk of CVD emergency department visits were associated with acute increases in PM$_{2.5}$. |
| Peralta, 2020        | Ventricular                                                                  | PM$_{2.5}$ | An acute increase in PM$_{2.5}$ was associated with 39% higher                                          |
| Reference | Sample Size/Study Details | Exposure | Effect | Notes |
|-----------|---------------------------|----------|--------|-------|
| Hu, 2020  | 4,720 acute MI emergency hospitalizations | PM$_{0.01-0.03}$, PM$_{0.03-0.05}$, PM$_{0.05-0.10}$, PM$_{0.10-0.30}$ | For an IQR increase of particle number concentrations for size ranges 0.01-0.03 μm, 0.03-0.05 μm, 0.05-0.10 μm, and 0.10-0.30 μm, acute MI hospitalizations increased by 6.68% (95% CI 2.77-10.74), 6.53% (95% CI 2.08-11.17), 5.78% (95% CI 0.92-10.88%), and 5.92% (95% CI 1.31-10.74), respectively. |
| Kuźma, 2020 | 2,645 acute coronary syndrome hospital admissions | NO$_2$ | The increase in the number of acute coronary syndrome hospitalizations was associated with an IQR increase in NO$_2$, with an OR of 1.08 (95% CI 1.02-1.15), 1.09 (95% CI 1.01-1.18), and 1.11 (95% CI 1.00-1.22) for patients with acute coronary syndrome, non-ST-segment elevation MI, and unstable angina, respectively. |
| Wang, 2020  | 45,714 CVD hospitalizations | PM$_{2.5}$, PM$_{10}$ | A 10 µg/m$^3$ acute increase in PM$_{2.5}$ and PM$_{10}$ contributed to a 1.01% (95% CI 0.67-1.34) and 0.48% (95% CI 0.26-0.70) increase in CVD hospitalizations, respectively. |
| So, 2020  | 24,541 females | PM$_{2.5}$, PM$_{10}$ | PM$_{2.5}$ was associated with CVD mortality (HR 1.14, 95% CI 1.03-1.26 per IQR increase). A similar association was found for PM$_{10}$. |
| Phosri, 2020 | Citywide CV hospital admissions | PM$_{10}$ | Acute changes in PM$_{10}$ were associated with increased risk of CV hospital admissions. |
| Mueller, 2020 | 7,752 IHD and 14,228 CBVD hospital visits | PM$_{10}$ | IRRs for acute increases in PM$_{10}$ and outpatient visits were 1.020 for CBVD (95% CI 1.004-1.035) and 0.994 (95% CI 0.974-1.014) for IHD. |
| Author, Year | Study Design | PM<sub>2.5</sub> Constituents | Findings |
|-------------|-------------|-------------------------------|----------|
| Yang, 2020  | Nationwide study (161 communities) | PM<sub>2.5</sub> constituents | Acute changes in elemental carbon, nitrate, organic carbon, ammonium, and sulfate were related to risk of CV and MI mortality. |
| Rodins, 2020| 4,105 subjects | NO<sub>2</sub>, PM<sub>10</sub>, PM<sub>2.5</sub> | No association between air pollutants and risk of CHD was observed. |
| Hystad, 2020| 157,436 subjects | PM<sub>2.5</sub> | A 10 μg/m<sup>3</sup> increase in PM<sub>2.5</sub> was associated with increased risk of CVD events (HR 1.05, 95% CI 1.03-1.07), MI (HR 1.03, 95% CI 1.00-1.05), stroke (HR 1.07, 95% CI 1.04-1.10), and CVD mortality (HR 1.03, 95% CI 1.00-1.05). |
| Wang, 2020  | 56,827 CV emergency ambulance dispatches | PM<sub>2.5</sub>, PM<sub>2.5-10</sub>, PM<sub>10</sub> | A 10 μg/m<sup>3</sup> increase in PM<sub>2.5</sub>, PM<sub>2.5-10</sub>, and PM<sub>10</sub> was associated with an increase of 0.69% (95% CI 0.00-1.39), 2.04% (95% CI 0.64-3.45), and 0.60% (95% CI 0.11-1.10) in CV emergency ambulance dispatches. |
| Tapia, 2020 | 86,970 cardiorespiratory deaths | PM<sub>2.5</sub> | An acute increase per 10 μg/m<sup>3</sup> in PM<sub>2.5</sub> was associated with cardiorespiratory mortality (RR 1.029, 95% CI 1.01-1.05). |
| Cramer, 2020| 22,882 females | PM<sub>2.5</sub>, PM<sub>10</sub> | PM<sub>2.5</sub> (HR 1.35, 95% CI 1.01-1.81) and PM<sub>10</sub> (HR 1.35, 95% CI 1.01-1.81) was associated with increased risk of fatal MI. |
| Dahlquist, 2020| 8,899 subjects | PM<sub>2.5</sub> | Acute increases in PM<sub>2.5</sub> increased the risk of AF. |
| Li, 2020    | 118,229 subjects | PM<sub>2.5</sub> | Per 10 μg/m<sup>3</sup> increase in PM<sub>2.5</sub>, for total CHD the HR was 1.43 (95% CI 1.35-1.51), for nonfatal CHD the HR was 1.45 (95% CI 1.36-1.56), and for fatal CHD the HR was 1.38 (95% CI 1.25-1.53). |
| Liang, 2020 | 116,972 subjects | PM<sub>2.5</sub> | Per 10 μg/m<sup>3</sup> increase in PM<sub>2.5</sub>, the HRs were 1.251 (95% CI 1.220-1.283) for CVD incidence and 1.164 (95% CI 1.117-1.213) for CVD mortality. |
| Gestro, 2020| 1,625 emergency department admissions for acute coronary | PM<sub>2.5</sub> | Acute increases in PM<sub>2.5</sub> increased the risk of emergency department admissions for acute coronary syndrome. |
| Study          | Number                  | Exposure | Effect of Exposure |
|---------------|-------------------------|----------|--------------------|
| Kim, 2020     | 38,928 OHCA             | PM$_{2.5}$ | Acute changes in PM$_{2.5}$ was associated with a higher risk of OHCA (1.59%, 95% CI 1.51-1.66 per 10 μg/m$^3$ increase). |
| Yang, 2020    | 1,016,579 outpatients   | PM$_{2.5}$ | An increase per 10 μg/m$^3$ in PM$_{2.5}$ was associated with a 0.584% (95% CI 0.346-0.689) increase in cardiac arrhythmias. |
| Choi, 2020    | 40,899 cancer survivors | PM$_{2.5}$ | PM$_{2.5}$ was associated with greater risk of CVD (HR 1.31, 95% CI 1.07-1.59). |
| Kojima, 2020  | 103,189 OHCA            | PM$_{2.5}$ | A 10 μg/m$^3$ increase in PM$_{2.5}$ was associated with a 1.6% increase in OHCA. |
| Yan, 2020     | 37,386 subjects         | SO$_2$    | HR of hypertension incidence for a 10 μg/m$^3$ increase in SO$_2$ was 1.176 (95% CI 1.163-1.189). |
| Yang, 2020    | 116,821 subjects        | PM$_{2.5}$ | A 10 μg/m$^3$ increase in PM$_{2.5}$ resulted in a HR of 1.22 (95% CI 1.16-1.27) for cardiometabolic mortality. |
| Wang, 2020    | 53 million Medicare beneficiaries | PM$_{2.5}$ | A 10 μg/m$^3$ increase in PM$_{2.5}$ was associated with an 8.8% increase in CVD deaths. |
| Ishii, 2020   | 137,678 acute MI cases  | PM$_{2.5}$ | Acute increases per 10 μg/m$^3$ in PM$_{2.5}$ were associated with admission for acute MI, MI with nonobstructive coronary arteries, and MI with CAD. |
| Liu, 2020     | 306,963 CVD hospital admissions | PM$_{2.5}$, PM$_{10}$, SO$_2$, NO$_2$ | CVD hospitalizations were increased by 0.401% (95% CI 0.029-0.775), 0.316% (95% CI 0.086-0.547), 0.903% (95% CI 0.252-1.559), and 2.647% (95% CI 1.607-3.697) per 10 μg/m$^3$ acute increase in PM$_{2.5}$, PM$_{10}$, SO$_2$, and NO$_2$, respectively. |
| Zhao, 2020    | 249,372 OHCA            | PM$_{2.5}$, CO, O$_{3}$, SO$_2$ | PM$_{2.5}$, CO, O$_{3}$, and, SO$_2$ were associated with OHCA. |
| Qiu, 2020     | hospital admissions due to acute MI (156,717), congestive HF | PM$_{2.5}$ | A 10 μg/m$^3$ acute increase in PM$_{2.5}$ exposure was associated with an increase of 4.3% (95% CI 2.2-6.4) in acute MI hospital admission rate, 3.9% (95% CI 2.4-5.5) in congestive HF rate, and 2.6% (95% CI 0.4-4.7) in ischemic stroke rate. |
| Study            | Number of Subjects/Events | Exposure | Effect Size | Reference |
|------------------|---------------------------|----------|-------------|-----------|
| Kim, 2020        | 436,933 subjects          | PM$_{2.5}$ | An increase per 10 μg/m$^3$ in PM$_{2.5}$ increased CV mortality by 4.7% (95% CI 3.6-5.8). | 169       |
| Chen, 2020       | 1,335 cases of acute aortic dissections | PM$_{2.5}$ | A 10 μg/m$^3$ acute increase in PM$_{2.5}$ was associated with a 3.38% (95% CI 1.02-5.79) increase in acute aortic dissection hospitalizations. | 170       |
| Zhu, 2020        | Citywide CV hospitalizations | Combination of PM$_{2.5}$, PM$_{coarse}$, CO, SO$_{2}$, NO$_{2}$, O$_{3}$ | An acute increase per 10 μg/m$^3$ in multiple air pollutants increased the emergency department visits for CV causes by 4.36% (95% CI 1.06-7.76). | 171       |
| Nhung, 2020      | 135,101 hospital records | PM$_{2.5}$ | Acute increases in PM$_{2.5}$ per IQR increase were associated with a 6.3% (95% CI 3.0-9.8) increase in admissions for IHD and 23.2% (95% CI 11.1-36.5) for HF. | 172       |
| Lefler, 2019     | 635,539 subjects          | PM$_{2.5}$, PM$_{2.5-10}$, SO$_{2}$ | PM$_{2.5}$, PM$_{2.5-10}$, and SO$_{2}$ were associated with increased risk of cardiopulmonary and all-cause mortality. | 173       |
| Wei, 2019        | 95,277,169 hospital admissions | PM$_{2.5}$ | Acute increases in PM$_{2.5}$ were associated with increased CV hospital admissions. | 174       |
| Lee, 2019        | 670 patients hospitalized with the first onset of AF | PM$_{2.5}$ | The occurrence of AF was associated with acute increases in PM$_{2.5}$. | 175       |
| Lim, 2019        | 548,780 subjects          | O$_{3}$ | O$_{3}$ was associated with deaths caused by CVD (HR 1.03, 95% CI 1.01-1.06 per 10 ppb increase) and IHD (HR 1.06, 95% CI 1.02-1.09). | 176       |
| Weaver, 2019     | 2,192 cardiac catheterization patients | PM$_{2.5}$ | PM$_{2.5}$ was related to CAD, MI, and hypertension. | 177       |

PM$_{(diameter\ size)}$: Particulate matter, OR: Odds ratio, CAD: Coronary artery disease, CI: Confidence interval, IQR: Interquartile range, MI: Myocardial infarction, BC: Black carbon, CO: Carbon monoxide, SO$_{2}$: Sulfur oxides, CVD: Cardiovascular disease, SBP: Systolic blood pressure, DBP: Diastolic blood pressure, HF: Heart failure, HR: Hazard ratio, CHD: Coronary heart disease, SO$_{2}$: Sulfur dioxide, IHD:
Ischemic heart disease, NO₂: Nitrogen dioxide, RR: Relative risk, COPD: Chronic obstructive pulmonary disease, O₃: Ozone, AF: Atrial fibrillation, NOₓ: Nitrogen oxides, CBVD: Cerebrovascular disease, IRR: Incidence rate ratio, OHCA: Out-of-hospital cardiac arrest, Oₓ: Photochemical oxidants

Suppl. Table S5. Animal in vivo studies on association of cardiovascular and endothelial dysfunction, inflammation, or oxidative stress with air pollution. Articles before 2013 are reviewed in ¹⁷⁸, ¹⁷⁹. Only articles that are not mentioned in the main article text are listed here.

| First author / year | Animals and model | Air pollutants | Major outcomes                                                                                                                                                                                                 | Ref   |
|---------------------|-------------------|----------------|---------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------|-------|
| Ying, 2013          | Wildtype and ApoE⁻/⁻ mice | Whole body concentrated ambient PM₂.₅ (68 µg/m³) and/or Ni (450 ng/m³) for 2 weeks                                                                                                                        | A strong inflammatory response (TNF-α, IL-6, MCP-1, E-selectin, VCAM-1) in mice exposed to concentrated ambient PM₂.₅ was observed. Ni exposure led to altered endothelial function (ACh) | ¹⁸⁰   |
| Study | Species | Treatment | Exposure | Outcome |
|-------|---------|-----------|----------|---------|
| Robertson, 2013 | C57BL/6 wildtype and CD36−/− mice | Filtered air (FA) or 1 ppm O₃ for 4h. | 6 h/d, 5 d/w | Compared to identical exposure to FA in WT mice, O₃ induced a reduction (by 85%) in Ach dependent relaxation. CD36−/− mice were robust against the O₃-induced alterations of Ach-dependent vasorelaxation in aortic rings. When compared with serum from WT mice exposed to a single dose of ozone, ex-vivo incubation of WT aortic rings with serum from CD36−/− mice exposed to ozone induced the same degree of vasodilatory impairment. |
| Chen, 2013 | Wildtype and ApoE−/− mice | Ambient particulate matter (PM) or filtered air continuously for 2 months | Diesel exhaust particulate exposure increased total cholesterol, LDL, TNF-alpha and C-reactive protein as well as TNF-alpha and IL-6 in bronchoalveolar lavage. Analysis of aortic arch indicated plaque area in PM-exposed group increased significantly compared to filtered air group. |
| Miller, 2013 | Wildtype and ApoE−/− mice | 4 weeks of twice weekly oropharyngeal instillation of 35 μL diesel exhaust particulate or saline | In ApoE−/− mice treated with diesel exhaust particles, brachiocephalic atherosclerotic plaques were larger and had more plaques per section of artery and buried fibrous layers. |
| Brocato, 2014 | FVB/N mice | 100 μg PM₁₀ (collected from Jeddah, Saudi Arabia) or water by aspiration | Increases in neutrophil concentration, TNF-alpha, and IL-6 levels were observed in mice exposed to PM₁₀. PM₁₀ induced genes involved in cholesterol and lipid metabolism, inflammation, and atherosclerosis. |
| Paffett, 2015 | Male Sprague-Dawley rats | Ozone (1 ppm) for 4h | Augmentations in broncho-alveolar lavage cellularity and neutrophil count and numbers of circulating neutrophils and macrophages in response to ozone exposure were observed. In rats exposed to ozone, the baseline coronary artery internal diameter was decreased and the percent increase in tone following isolation and mounting was elevated in the vessels. Likewise, in the ozone group coronary artery constriction in response to serotonin was more pronounced. Furthermore, ozone... |
| Study       | Species/Model     | Exposure/Condition                                      | Summary                                                                                                                                                                                                 |
|-------------|-------------------|----------------------------------------------------------|---------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------|
| Vella, 2015 | Wistar rats       | Ozone (0.8 ppm for 16 h)                                 | Insulin resistance was triggered by ozone exposure through muscle c-Jun N-terminal kinase activation, a process that was mirrored by toxic mediators in bronchoalveolar lavage fluids. Insulin resistance was prevented by pretreatment with the chemical chaperone 4-phenylbutyric acid, the antioxidant N-acetylcysteine, or the JNK inhibitor SP600125, indicating that ozone sequentially triggered oxidative stress, ER stress, and JNK activation leading to impaired insulin signaling in muscle. These data suggest that ozone could boost the development of diabetes. |
| Cui, 2015   | C57BL/6 mice      | PM$_4$ (10 µg) 3x per week for 1 month                  | PM administration decreased the levels of bone marrow-derived stem cells accompanied by increased ROS formation, impaired Akt activation, increased inflammation (TNF-α, IL-1β) and proliferation of stem cells (with or without induction of apoptosis – see the different results in the 2 references). Antioxidant treatment with N-acetylcysteine treatment or transgenic overexpression of 3 antioxidant enzymes (SOD1, SOD3, GPx-1) lead to reduced ROS formation, inflammatory mediators and prevented PM-triggered decrease in stem cell levels by restored Akt activity. |
| Wei, 2016   | Pregnant Sprague Dawley rats | Ambient traffic air pollutants (field experiment in Beijing with cages placed at a main road: PM$_{2.5}$ was 73.5 µg/m$^3$ over 2 weeks) | The risks of obesity and metabolic syndrome in rats and their offspring were increased by chronic exposure to air pollution particles. In dams and offspring chronic exposure to unfiltered traffic air pollutants increased tissue and systemic oxidative stress, increased perivascular and peribronchial inflammation in the lungs, induced dyslipidemia, and led to a pronounced proinflammatory status of epididymal fat. These results suggested that TLR2/4-dependent inflammatory activation and lipid oxidation |
in the lung can spill over systemically, resulting in inherited metabolic dysfunction and weight gain.

| Study | Genotype | Treatment | Observations |
|-------|-----------|-----------|--------------|
| Haberzettl, 2016 | C57Bl/6 wildtype and lung-specific ecSOD<sup>tg</sup> mice | Whole body concentrated ambient PM<sub>2.5</sub> or filtered air for 9 or 30 days. | 9-day PM<sub>2.5</sub> exposure was shown to suppress insulin-stimulated Akt and eNOS phosphorylation and to decrease IκBα (inhibitor of the transcription factor NF-κB levels in the aorta) in control diet-fed mice. Treatment with the antioxidant 4-hydroxy-2,2,6,6-tetramethylpiperidine-1-oxyl (TEMPOL) or lung-specific overexpression of ecSOD prevented PM<sub>2.5</sub>-induced vascular insulin resistance and inflammation. |
| Zhong, 2016 | Diabetes-prone KK mice | Ozone (0.5 ppm for 4 h/d, 5 d/week for 3 weeks) | O₃ exposure in KK mice induced an impaired insulin response as indicated by decreased plasma insulin and leptin levels. Chronic O₃ exposure was associated with lung inflammation and increased monocytes/macrophages in both blood and visceral adipose tissue with more inflammatory monocytes/macrophages at the systemic and local level along with higher CD4+ T cell activation. Multiple inflammatory genes including CXCL-11, IFN-γ, TNF-α, IL-12, and iNOS as well as oxidative stress-related genes such as Cox4, Cox5a, Scd1, Nrf1, and Nrf2 were up-regulated in visceral adipose tissue of mice exposed to O₃. |
| Orona, 2016 | BALB/c mice | UAP-BA and ROFA via intranasal instillation (1 mg/kg) | Local and systemic inflammatory responses were induced by acute exposure to Buenos Aires air particles (UAP-BA) and Residual Oil Fly Ash (ROFA) in middle-aged mice. This was evident by reduced alveolar area in the lung, epicard inflammation in the heart, higher IL-6 levels, and reduced paraoxonase 1 expression in serum as well as elevated systemic oxidative stress markers. |
| Wang, 2018 | Wildtype and AMPKα2<sup>−/−</sup> mice | PM<sub>2.5</sub> (64 µg/m³ for 6 months) | AMPKα2 deficiency exacerbated chronic PM<sub>2.5</sub> exposure-induced cardiac dysfunction and lung injury as mirrored by severe fibrotic lung injury and left ventricular dysfunction in AMPKα2<sup>−/−</sup> mice as compared to wildtype mice. Lungs and heart of AMPKα2<sup>−/−</sup> mice exposed to PM<sub>2.5</sub> showed lower levels of peroxiredoxin 5 and |
| Reference | Species | Exposure | Findings |
|-----------|---------|----------|----------|
| Guan, 2018 | Sprague-Dawley rats | PM$_{2.5}$ or filtered air for 6 or 12 weeks | Middle cerebral artery (MCA) narrowing and thickening in relation with pronounced expression of inflammatory cytokines were induced by PM$_{2.5}$ exposure for 12, but not 6 weeks. Omega-3 fatty acids significantly attenuated vascular alterations and inflammatory cytokine expression, without favorable changes in lipid profiles. |
| Rao, 2019 | Wildtype and brain-specific Adra$_{2b}$tg mice | Concentrated ambient PM$_{2.5}$ (8 to 10-fold enrichment of ambient air; 6 h/d, 5 d/week, for 12 weeks) | Fine ambient particulate matter (PM$_{2.5}$) induced behavior changes and increased blood pressure synergistically in high salt diet treated mice via upregulation of the α2B-adrenergic receptor in the brain of wildtype mice. Inflammatory genes (TLR2, TLR4 and IL-6) as well as antioxidant genes (SOD1, NQO1, Nrf2 and Gclm) were induced in the brain of PM$_{2.5}$-exposed wildtype mice. Brain-specific overexpression of the α2B-adrenergic receptor further aggravated the adverse effects of PM$_{2.5}$. |
| Gao, 2020 | Wildtype and AMPKα2$^{-/-}$ mice | PM$_{2.5}$ (10 mg/kg/d via intratracheal instillation) for 4 weeks, metformin (300 mg/kg/d) | PM$_{2.5}$-induced lung injury and cardiac dysfunction were prevented by metformin independent of AMP-activated protein kinase α2. In both the wildtype and AMPKα2$^{-/-}$ mice, metformin also decreased systemic and pulmonary inflammation, suppressed induction of pulmonary and myocardial fibrosis and oxidative stress, preserved left ventricular ejection fraction, and increased levels of mitochondrial antioxidant enzymes. |
| Hill, 2021 | Wildtype and lung-specific ecSOD$^{tg}$ mice | Concentrated ambient PM$_{2.5}$ (60 or 100 µg/m$^3$ for 9 d) | Fine particulate matter (PM$_{2.5}$) inhalation caused alterations in the plasma lipidome in wildtype mice leading to vascular inflammation and insulin resistance. PM$_{2.5}$ increased plasma levels of palmitate, myristate and palmitoleate but reduced a number of phospholipids. These PM2.5-induced changes were prevented in lung-specific ecSOD$^{tg}$ mice. |
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