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Effects of transportation noise and particulate matter on the cardiovascular system: What is the new evidence?

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ABSTRACT. Several decades of research provides compelling evidence that exposure to air pollution causes various diseases. Time series studies in Europe and the USA examined the short term effects on mortality and found about 1% increase for cardiovascular mortality per 10 µg/m^3 increase in daily PM2.5 concentration. A comprehensive analysis of 12 European cohort studies addressing the long term effects found a 13% increased risk of coronary events per 5 µg/m^3 increase in estimated annual mean PM2.5. Noise exposure triggers an increase in sympathetic responses (fight-flight reactions) and increased release of corticoids (defeat reactions). In 2018 the World Health Organization has published new Environmental Noise Guidelines reporting an 8% increase in risk of incident ischemic heart disease per 10 dB(A) increase in road traffic noise exposure. Transportation noise and air pollution impact health through different pathways, though they also share many biologic pathways. In a recent cohort study, we did not find indications for synergistic or antagonistic effects from combined exposure to noise and air pollution. A study estimating the Environmental Burden of Disease in Europe concluded that ambient air pollution and transportation noise are the two most relevant environmental stressors for the population living in Europe.

Key words: *ultrafine particles, cardiovascular diseases, noise.*

RIASSUNTO. Anni di ricerca hanno confermato che l'esposizione a inquinanti ambientali causa molte patologie. Gli studi longitudinali in Europa e USA che hanno esaminato gli effetti a breve termine hanno evidenziato un aumento di circa l'1% della mortalità cardiovascolare ogni 10 µg/m^3 di incremento giornaliero della concentrazione di PM2.5. Una analisi comprensiva di 12 coorti Europee, che hanno valutato gli effetti a lungo termine, ha trovato un aumento del 13% di eventi coronarici ogni 5 µg/m^3 di incremento delle PM2.5 su base annuale. L'esposizione a rumore stimola un aumento della riposta simpatica (reazioni di attacco) e un aumentato rilascio di corticosteroidi (reazioni di difesa). Nel 2018 l'Organizzazione Mondiale della Sanità ha pubblicato le nuove line guida che riportano un aumento dell'8% dei casi incidenti di patologie ischemiche cardiache ogni 10 dB(A) di aumento al rumore del traffico stradale. Il rumore del traffico e l'inquinamento ambientale impattano la salute attraverso meccanismi diversi, ma hanno anche alcuni effetti biologici comuni. In uno studio di coorte recente, non abbiamo trovato indicazioni a supporto di un effetto sinergico o antagonista dall'esposizione combinata a rumore e inquinamento ambientale. Uno studio che ha valutato il carico ambientale delle malattie in Europa ha concluso che l'inquinamento ambientale e il rumore veicolare sono i due più importanti fattori di rischio per la popolazione che vive in Europa.

Parole chiave: particelle ultrafini, rumore, patologie cardiovascolari.

Air pollution and cardiovascular diseases

Ambient air pollutants include both, gaseous contaminants and particulate matter (PM) of different size fractions such as PM10 (diameter <10 µm), PM2.5 (<2.5 µm) or ultrafine particles (<0.1 µm). The smaller the particles and the less water soluble, the deeper they penetrate into the lung. PM is either primary or secondary in origin and is generated naturally (pollen, spores, salt spray, and soil erosion) and by human activities like agriculture, industrial process, traffic, heating or construction work.

Several decades of research provides compelling evidence that exposure to air pollution causes various diseases (Figure 1) (1). Most studies looked into the health effects of PM10 and PM2.5 but there is also growing evidence about health effects from ultrafine particles (2).

Time series studies in Europe and the USA examined the *short term effects* on mortality and demonstrated increases in the cardiovascular mortality rate of about 1% per 10 µg/m^3 increase in daily PM2.5 concentrations. Depending on the constituents of PM, effect size may vary. There are some indications of stronger associations for elemental carbon but effect estimates show a high variability across cities (3).

Cohort studies addressing the *long term effects* on cardiovascular mortality report higher effects than time series study. A recent analysis of 12 European cohort studies with 100'000 people enrolled from 1997 to 2007 found a 13% increased risk of coronary events per 5 µg/m^3 increase in estimated annual mean PM2.5 and a 12% increased risk of coronary events per 10 µg/m^3 increase in estimated annual mean PM10 (4).

Transportation noise and cardiovascular diseases

Very high noise levels (> 85 dBA) is leading to hearing loss due to its direct damaging effects on hair cells of the inner ear (5). However, for negative effects on the cardiovascular system other pathways are relevant, which involves the cognitive perception and the physiological reaction to noise exposure at lower levels. In this context noise is defined as unwanted sound. Noise exposure triggers an increase in sympathetic responses (fight-flight re-

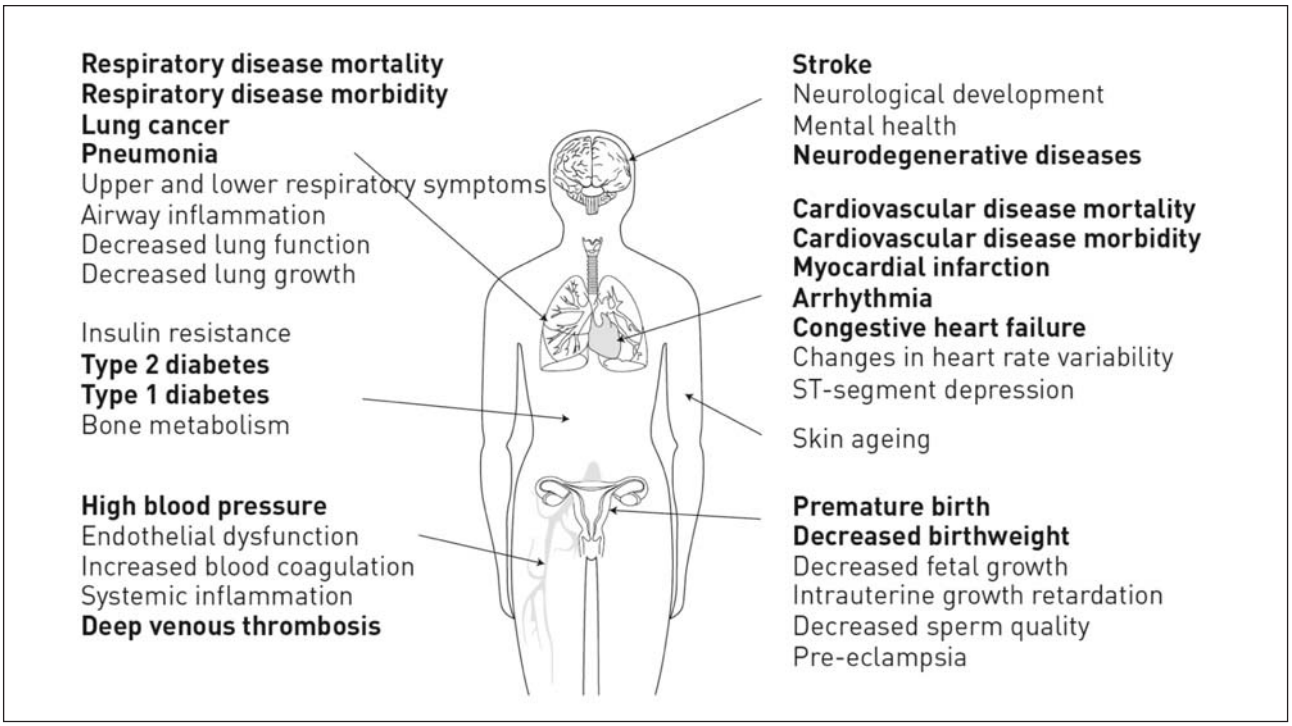

Figure 1. ***Overview of effects from ambient air pollution from Thurston et al, 2017 (1). Bold type indicates conditions currently included in the Global Burden of Disease categories***

actions) and increased release of corticoids (defeat reactions) resulting in an increased blood viscosity, activation of blood coagulation and an increased blood pressure (6). If noise stress persists for many years cardiovascular diseases may develop. Notably, the cognitive perception of noise is not a prerequisite for its adverse cardiovascular effects (7).

In 2018 the World Health Organization (WHO) has published new Environmental Noise Guidelines including systematic reviews for various diseases (7). For incident ischemic heart disease (IHD), road traffic noise was found to be significantly associated with an 8% increase in risk per 10 dB(A) increase in noise exposure (10). Using the GRADE (Grading of Recommendations, Assessment, Development and Evaluations) approach, the WHO expert group ranked the quality of evidence for an effect of road traffic noise on IHD as high. For rail and aircraft noise the quality of evidence was ranked lower due to fewer studies of high quality. The WHO recommended L_{den} (equivalent noise level over the whole day) guidelines of 53 dB for road traffic, 54 dB for railway noise and 45 dB for aircraft noise to prevent adverse health effects. To prevent effects on sleep, the WHO recommended nighttime noise levels of 45 dB for road traffic, 44 dB for railway noise and 40 dB for aircraft noise. Since the data collection of the WHO report has ended in August 2015, various high quality cohort studies have led to further support of an association between traffic noise and cardiovascular diseases.In Switzerland, we conducted a nationwide cohort study on cardiovascular deaths between 2000 and 2008 and modeled road traffic, railway and aircraft noise levels at each address of the 4.4 million adults aged >30 years in the Swiss National Cohort (SNC). After adjusting for relevant confounders significant exposure response associations were found for various cardiovascular causes of deaths (Table I). The study also indicated that characteristic of the noise source is also important. Highly fluctuating noise exposure during the night (e.g. from trains and planes) was more strongly associated with acute coronary events, whereas for chronic cardiovascular diseases continuous-like daytime noise exposure (e.g. from a highway) was most relevant (9).

Joint effects of air pollution and cardiovascular diseases

Transportation noise and air pollution impact health through different pathways, though they also share many biologic pathways (10). This raises the questions about potential synergistic or antagonistic effects from combined exposure to noise and air pollution. Further, mutual confounding is also of concern, since transportation noise and air pollution both originate from traffic and correlations between long-term traffic noise and air pollution ranging from 0.16 to 0.72 have been reported in the literature (11). A systematic review of nine studies comprising outcomes such as hospital discharge registers, self-reported medication intake, and mortality found that less than 10% of the effect estimate of noise was attenuated after adjustment for air pollution or vice versa (11). and thus concluded that confounding of cardiovascular effects by noise or air pollution is low. However, the authors concluded that improvements in exposure assessment may change the situation.

In our SNC study we have thus looked closer into this topic by conducting small-scale spatial modelling for

Table I. ***Relative risk per 10 dB increase in noise levels for various cardiovascular causes of death from Héritier et al, 2017 (8). Significant associations are printed in bold***

Cause of death	Source	Excess Risk (%)	95% Confidence intervals	
All cardiovascular diseases	L_{den} Road	**2.5**	**1.8**	**3.2**
	L_{den} Railway	**0.5**	**0.0**	**1.0**
	L_{den} Aircraft	-0.6	-0.15	0.2
Ischemic heart diseases	L_{den} Road	**2.3**	**1.2**	**3.4**
	L_{den} Railway	1.2	0.5	2.0
	L_{den} Aircraft	-0.9	-2.2	0.3
Mycordial infarction	L_{den} Road	**4.0**	**2.1**	**5.9**
	L_{den} Railway	**2.0**	**0.7**	**3.3**
	L_{den} Aircraft	**2.7**	**0.6**	**4.3**
Hypertension related causes	L_{den} Road	**5.3**	**3.0**	**7.5**
	L_{den} Railway	1.1	-0.5	2.7
	L_{den} Aircraft	1.2	-1.5	3.9
Heart Failure	L_{den} Road	**5.1**	**2.7**	**7.4**
	L_{den} Railway	-0.3	-2.0	1.4
	L_{den} Aircraft	5.6	2.8	8.5
Stroke	L_{den} Road	1.1	-0.7	2.8
	L_{den} Railway	-0.5	-1.7	0.8
	L_{den} Aircraft	-0.9	-4.9	3.2

* Multi exposure models adjusted for sex, neighborhood index of socio-economic position, civil tatus, education level, mother tongue, nationality and NO2 exposure

noise and air pollution (NO2 and PM2.5). Overall, we did not find any indications for synergistic or antagonistic effects on myocardial infarction for combined exposure to both types of pollutants. We observed that myocardial infarction was associated with transportation noise, independent from air pollution exposure. Conversely, associations between air pollution and myocardial infarction became smaller after adjustment for noise exposure, in particular in terms of NO2. The study thus suggests that air pollution studies not adequately adjusting for transportation noise exposure may overestimate the cardiovascular disease burden of air pollution. This finding need to be confirmed in future studies applying small scale exposure modelling.

Global burden of air pollution and transportation noise

In 2017, the Global Burden of Disease Risk Factor Collaborators estimated 4.9 million deaths and 147 million disability adjusted life years (DALYs) attributable to indoor and outdoor air pollution on the whole globe (12). A substantial part was attributable to ischemic heart diseases from ambient air pollution (1 million deaths, 22 million DALY's). Occupational noise is estimated to cause 6 million DALY's due to hearing loss. Strikingly, health impact of environmental noise is not included in the Global Burden of Diseases report, possibly reflecting lack of noise exposure data for some parts of the world, which would be needed for these calculations. However, the European Office of the WHO concluded that traffic-related noise accounts for over 1 million DALYs each year in the European Region (13). A study estimating the Environmental Burden of Disease in Europe for nine risk factors concluded that ambient air pollution and transportation noise are the two most relevant environmental stressors for the population living in Europe (14).

In the last decades awareness for detrimental effects from air pollution has substantially increased all over the world. However, awareness for negative health effects from environmental noise is not yet at the same level. It is thus important to better communicate the substantial public health impact from noise on a global scale, while keeping the awareness for health effects from air pollution.

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