Prevention

Environmental stressors and cardio-metabolic disease: part I—epidemiologic evidence supporting a role for noise and air pollution and effects of mitigation strategies

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Traffic noise and air pollution together represent the two most important environmental risk factors in urbanized societies. The first of this two-part review discusses the epidemiologic evidence in support of the existence of an association between these risk factors with cardiovascular and metabolic disease. While independent effects of these risk factors have now clearly been shown, recent studies also suggest that the two exposures may interact with each other and with traditional risk factors such as hypertension and type 2 diabetes. From a societal and policy perspective, the health effects of both air pollution and traffic noise are observed for exposures well below the thresholds currently accepted as being safe. Current gaps in knowledge, effects of intervention and their impact on cardiovascular disease, will be discussed in the last section of this review. Increased awareness of the societal burden posed by these novel risk factors and acknowledgement in traditional risk factor guidelines may intensify the efforts required for effective legislation to reduce air pollution and noise.

Introduction

According to the 'Environmental Burden of Disease in European Countries' project report, particulate matter air pollution, together with traffic noise pollution, contribute to >75% of the burden of disease attributable to environmental factors.1 With increasing urbanization, the degree to which people are exposed simultaneously to these factors is likely to further increase in the future, particularly in developing countries. Currently, 17 megalities in Asia rank among the top 30 cities in terms of premature cardiovascular mortality and account for 7% of the global burden of disease attributable to outdoor air pollution alone.2 In the first part of this series on environmental stressors and disease, we will focus on the epidemiology of cardiometabolic disease mediated by air and traffic noise pollution, the strength of the findings thus far, and discuss the effects of mitigation strategies in improving cardiovascular outcomes. The main topic of the ESC congress 2015 in London was the environment and its impact on cardiovascular disease (CVD). In the ESC congress news 2015, the question ‘Looking at the crystal ball: What will change the future of cardiology’ was addressed. In his article, Valentin Fuster predicted that there will be a strong shift in focus to identification and prevention of environmental causes of CVD (http://www.nxtbook.com/nxtbooks/md_conference_express/esccongress2015/).

In the present review, the epidemiology of air and noise pollution, areas of current knowledge gaps and impact of intervention will be discussed.
Global burden of air and noise pollution

Household air pollution and ambient outdoor air pollution are the 3rd and 9th leading risk factors contributing to global mortality and disability, accounting for 6.6 million deaths and 7.6% of global DALYs, of which 3.5% is from outdoor air pollution.\(^3\) More than 80% of the world’s population lives in areas in which particulate matter reaches or is above thresholds recommended by the World Health Organization.\(^4\) Similar to the pervasive influence of air pollution, noise, and its non-auditory effects are pervasive in the urban environment. The non-auditory effects including annoyance, sleep disturbance, and psychological stress are widely suspected to cause worldwide disability. According to the WHO, at least 1 million healthy life-years (disability-adjusted life-years) are lost every year in western European countries because of environmental noise,\(^5\) and cardiovascular (CV) disease contributes to the vast majority of these deaths. It is estimated that 40% of the population in EU countries is exposed to road traffic noise at levels exceeding 55 dB(A), and 20% exposed to levels exceeding 65 dB(A) during the daytime; >30% of the population is exposed to levels exceeding 55 dB(A) at night.\(^6\) Extreme levels of traffic noise pollution are reached in Asian urbanized environments, where a much higher proportion of the population may reach LDEN levels (Day–Evening–Night level, i.e. the average sound pressure level measured over a 24-h period) of 60–64 dB or more compared with European cities.\(^7\)

Noise and air pollutants: components, sources, and standards

Noise is any sound that is subjectively unpleasant and disturbing and causes unwanted effects through a direct (auditory, such as hearing loss) or indirect (disturbing sleep and communication and sympathetic arousal) pathway. The impact of noise on physiological functions and psychological processes depends on its characteristics, intensity, and nature. Noise pollution in most urban environments originates from transportation (land and air) but may also originate from loudspeakers, sirens, automobile horns, and machinery from industries. Measures of intensity of noise and sounds are reported in several recent reviews (Figure 1).\(^8\),\(^9\) Ambient air pollution is a complex mixture of thousands of particulate and gaseous components. Approximately 98% of the pollutant mass is directly attributable to gases or vapour-phase compounds such as CO, non-methane hydrocarbons or volatile organic carbons (VOCs), NO\(_2\), NO, O\(_3\), and SO\(_2\), all of which can have independent and potentially synergistic or antagonistic effects. Particulate matter (PM) is broadly categorized based on its

![Decibel scale (dB(A))]  | PM concentration (µg/m\(^3\)) | Particle size (µm)
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Threshold of pain  | 130 | > 2.5 m
Aircraft on take-off | 120 | 2.5 µm
Rockband | 110 | 1.0 µm
Jackhammer | 100 | 0.5–0.1 µm
Truck | 90 | 0.1–0.025 µm
Telephone ringing | 80 | 0.025–0.005 µm
Passenger car | 70 | 0.005–0.001 µm
Conversation | 60 | 0.001–0.0001 µm
Rain | 50 | 0.0001–0.00001 µm
Quiet living room | 40 | 0.00001–0.000001 µm
Whisper | 30 | 0.000001–0.0000001 µm
Tickling of a watch | 20 | 0.0000001–0.00000001 µm
Rustling leaves | 10 | 0.00000001–0.000000001 µm
Threshold of hearing | 0 | 0.000000001–0.0000000001 µm

WHO PM\(_{2.5}\) 24h standard | 65 | PM2.5, particulate matter >2.5 m; PM10, particulate matter >10 m. IT, interim targets.
WHO Interim targets (PM2.5) | 50 | WHO PM\(_{2.5}\) 24h standard
WHO PM\(_{2.5}\) Annual standard | 45 | WHO PM\(_{2.5}\) 24h standard
WHO PM\(_{10}\) Annual standard | 40 | WHO PM\(_{2.5}\) Annual standard
Healthy | 35 | WHO PM\(_{10}\) Annual standard
Unhealthy | 30 | WHO PM\(_{2.5}\) Interim targets (PM2.5)
WHO Interim targets (PM2.5) | 25 | WHO PM\(_{10}\) Annual standard
WHO PM\(_{10}\) Annual standard | 20 | WHO PM\(_{2.5}\) Interim targets (PM2.5)
WHO PM\(_{10}\) Interim targets | 15 | WHO PM\(_{10}\) Annual standard
WHO PM\(_{10}\) 24h standard | 10 | WHO PM\(_{10}\) Interim targets
WHO PM\(_{10}\) 24h standard | 5 | WHO PM\(_{10}\) 24h standard
WHO PM\(_{10}\) 24h standard | 0 | WHO PM\(_{10}\) 24h standard

LDEN: The 24 h LAeq with a 5 dB penalty for the evening (usually 6 p.m. to 10 p.m. or 7 p.m. to 11 p.m.) and a 10 dB penalty for the night (usually 10 p.m. to 6 a.m. or 11 p.m. to 7 a.m.). The penalties are introduced to indicate people’s extra sensitivity to noise during the evening and the night. With respect to long-term health effects, these metrics are usually calculated as average annual exposure indicators. PM2.5, particulate matter <2.5 m; PM10, particulate matter <10 m. IT, interim targets.
Spatial and temporal variation and correlation of air pollution and traffic noise levels

There is substantial spatial and temporal variation of air pollution and noise levels. Traffic-related pollutants and noise often peak during the late morning and evening rush hours, and show substantial changes even within short distances. Meteorological conditions such as atmospheric stability can significantly alter the horizontal propagation of both noise and air pollution and thus the size of the population exposed. Reflecting their origin from sources such as traffic, large urban-rural differences are found for primary combustion pollutants such as nitrogen oxides (NO and NO\textsubscript{2}) and particulate black carbon. The gradients for both noise and air pollutants are dependent upon meteorological conditions, including diurnal changes in vertical mixing height, wind speed, and temperature.\textsuperscript{16} In the case of noise, the gradients are substantial as the intensity of noise decreases exponentially with the distance from its source. The gradients for air pollution may also differ depending upon the pollutant. For example, ultrafine particle gradients are very steep while NO\textsubscript{2} gradients are more-gradual and PM\textsubscript{2.5} gradients are generally stable over long distances.

A common issue to consider, when assessing the CV health effects of traffic noise and air pollution exposure either alone or in conjunction, is that while regional levels (i.e. background air pollution or noise that most individuals more uniformly encounter) and proximity to the roadway generally affects the outdoor air quality of large segments of the population uniformly, personal exposure may demonstrate considerable variation, due to different indoor microenvironments (e.g. variable indoor sources of air pollution and penetration of outside air indoors) and individual activities (e.g. time in traffic and different indoors exposures). Studies that have measured both transportation noise and air pollution simultaneously, not surprisingly suggest that these measures are indeed correlated, although the degree of correlation may markedly vary: The highest correlation is typically found between traffic-related air pollutant measures such as black carbon or NO\textsubscript{2} and road traffic noise and is less so for regional air pollutants such as PM\textsubscript{2.5}. These variable results may reflect the nature of pollutants, as regional pollutants such as PM\textsubscript{2.5} may not be expected to correlate well with traffic noise while NO\textsubscript{2} and black carbon (depending on sources in the study) may be more correlated. Road traffic factors, such as the speed and traffic load, may also differentially affect noise and traffic-related air pollution. During traffic congestion, when traffic is at standstill or at lower engine speeds, noise levels may be lower but emissions are dramatically higher contributing to marked surges in traffic-related air pollutants. When traffic is moving well, noise levels may be higher but emissions may be lower.\textsuperscript{17} Environmental factors such as road conditions, noise barriers, and surrounding buildings are well known to influence traffic noise but are not expected to influence air pollution substantially. The volume of traffic could determine air pollution levels but do not affect noise levels as much. For example, when traffic volume doubles, traffic noise levels increase 3 dB(A).\textsuperscript{17} Finally, meteorological factors may have opposing effects on traffic noise and air pollution. Rain and wet road surfaces may increase road traffic noise but may decrease ambient air pollution. Wind direction and speed may additionally have powerful effects on traffic pollution levels.

The highly associated nature of traffic noise and air pollution makes it challenging to isolate their independent effects on cardiovascular events in epidemiological studies. A few studies have included both exposure when estimating associations with CVD, and attempted to assess the independent contribution of noise from air pollution and vice versa. The results are somewhat variable, with some studies demonstrating an independent effect of noise and/or air pollution on cardiovascular morbidity and mortality, while others find marked attenuation of effects after adjusting for the other (Supplementary material online, Table S1).\textsuperscript{18} Whether noise and air pollution have differing, additive, synergistic, and/or confounding effects upon CV health is still incompletely understood. Further, the degree to which measurement of personal co-exposures to both traffic noise and air pollution can provide additional information on mechanisms, isolate these effects from other variables such as temperature, diet and reduce exposure misclassification in epidemiological studies is an important area of investigation. Finally, both ambient air and noise pollution levels may potentially have an outsized impact in environments whereambient levels are high and adequate housing and/or insulation may not be available, such as in urban areas of warm climates or in developing countries that lack adequate infrastructure, insulated homes and/or air conditioning and bring large segments of the population in close and nearly continuous contact with high levels of noise and air pollution exposure. In such environments, exposure conditions may often be extreme with levels of exposure drastically differing from even the most congested environments in Europe and North America.

Epidemiological evidence of traffic noise and cardiometabolic disease

Although the strength of the associations varies significantly across studies, chronic exposure to road traffic, and/or railway or aircraft
noise is associated with elevated blood pressure, arterial hypertension, and increased use of antihypertensive medication. Supplementary material online, Table S3 depicts the studies that have demonstrated an association between noise levels and hypertension. Similarly, there are many studies that demonstrate a link between noise exposure and propensity to ischaemic heart disease, including fatal myocardial infarction and stroke. The odd’s ratios describing these relationships are in the range of 1.07 – 1.17 per 10 dB increase.\(^\text{8-10}\) (Figure 2) in adults, but also in children aged 9 – 10 years.\(^\text{26}\) The impact of traffic noise on CVD was analysed in two recent meta-analysis.\(^\text{10-19}\) Babisch et al. report a relative risk of 1.08 (95% confidence interval: 1.04, 1.13) per 10 dB(A) rise in weighted day-night noise levels within the exposure range of 52 – 77 dB(A). Different health outcomes of coronary health were considered, including the incidence of coronary artery disease or sudden cardiac death, if caused by myocardial infarction, acute myocardial infarction, mortality from coronary artery disease, and self-reported heart disease. Viennau et al. also evaluated the threshold and the shape of the exposure – response association.\(^\text{10}\) Based on the reference levels used in the studies included in their meta-analysis, the authors found that the association seemed to start as low as 50 dB, although they add that more studies are needed to further support research into the shape of this relationship.\(^\text{10}\)

Road traffic noise has also been linked to risk for stroke. In a population-based cohort of 57 053 people, Sorensen et al. found an HR of 1.14 for stroke (95% CI: 1.03 – 1.25) per 10 dB higher level of road traffic noise (\(L_{\text{den}}\)) in analyses adjusted for a number of potential confounders, including air pollution. There was a statistically significant interaction with age, with a strong association between road traffic noise and stroke among cases over 64.5 years (HR: 1.27; 95% CI: 1.13 – 1.43) and no association for those under 64.5 years (HR: 1.02; 95% CI: 0.91 – 1.14). The authors suggest that a generally more fragmented sleep structure with age renders elderly people more susceptible to sleep disturbances from noise. However, more studies into the effects of age on the relationship between noise and CVD are warranted.

These observations were supported by the findings of Halonen et al. who found that daytime road traffic noise at levels > 60 dB increased the risk of hospital admission for stroke with a relative risk of 1.05 (95% CI: 1.02 – 1.09) in adults, and 1.09 (95% CI: 1.04 – 1.14) in the elderly when compared with exposure to noise levels < 55 dB.\(^\text{31}\) Night time noise was associated with stroke admissions only among the elderly. Furthermore, daytime noise > 60 dB was significantly associated with all-cause mortality in adults [RR 1.04 (95% CI: 1.00 – 1.07)] and positive but non-significant associations were seen with mortality for ischaemic heart disease and stroke.

Traffic noise has been found to increase the risk of metabolic syndrome, obesity, and type 2 diabetes. In 2014, Eriksson et al. studied the effects of long-term aircraft noise exposure on body mass index, waist circumference, and type 2 diabetes. The authors found that an increase in 5 dB(A) was associated with a greater waist circumference of 1.5 cm. This observation fits with the concept that aircraft noise stimulates the release of cortisol which is known to contribute to central obesity.\(^\text{32-33}\) In 2015, four new studies were published adding to the growing evidence that traffic noise can increase the risk of adiposity. Two Danish studies using a large prospective cohort found exposure to road traffic noise to be associated with increases in BMI and waist circumference in both a cross-sectional and longitudinal design.\(^\text{34}\) Similarly, a Swedish cross-sectional study found a positive association between road traffic noise and waist circumference, but no association with BMI.\(^\text{35}\) In contrast, a Norwegian study found no association between road traffic noise and obesity markers, although the results indicated a positive association among highly noise sensitive women.\(^\text{37}\)

In 2013, a large Danish cohort study of 57 000 participants with 4000 cases of diabetes, found a 10-dB higher level of long-term

**Figure 2** (A) Exposure – response relationships of the associations between transportation noise and cardiovascular health outcomes. Data taken from Refs 10, 20, 27, 29 with permission from https://acoustics.org/cardiovascular-effects-of-noise-on-man-wolfgang-babisch. Note: Study-specific reference values were pooled after conversion to \(L_{\text{den}}\) using the derived meta-analysis weights of each study (according to Ref. 10) Road, road traffic noise; Air, aircraft noise; Hyp, hypertension; CHD, coronary heart disease. (B) Data for air pollution modified from cardiovascular mortality and exposure to airborne fine particulate matter and cigarette smoke: shape of the exposure – response relationship.\(^\text{30}\)
road traffic noise (5 years preceding diagnosis) to be associated with an increased risk of incident diabetes, with HR of 1.11 (95% CI: 1.05, 1.18) after adjusting for a number of potential confounders including lifestyle characteristics and air pollution (NOx). After applying a stricter definition of diabetes (2752 cases), the authors found an HR of 1.14 (95% CI: 1.06, 1.22) per 10-dB in 5-year mean road traffic noise.38 Similarly, a two-fold higher risk of type 2 diabetes (252 cases) was demonstrated for people exposed to intense road traffic in a nationwide German prospective cohort.39 Supplementary material online, Table S2 depicts the studies to date that have shown a relationship between traffic noise and diabetes mellitus and obesity/insulin resistance.

### Epidemiology of air pollution and cardiometabolic disease

A large epidemiologic literature provides compelling evidence that exposure to air pollution, especially combustion-related PM and associated co-pollutants contribute to overall CV mortality. The risk association in short-term time series analysis translates to a ≈ 1% elevation in CV-related mortality/10 μg/m³ of PM_{2.5} (≏ 1 SD increase in ambient levels in the USA and Europe).32 Importantly, as analyses based on lag periods up to 40 days, an even stronger association has been noted, indicating that the association with pollution is not the result of mortality harvesting (i.e., earlier death of patients whose short-term negative prognosis was already defined) alone. Recent studies have confirmed the association between air pollution and MI risk, particularly after long-term exposure. A meta-analytic review of long-term exposure and mortality indicated that a 10-μg/m³ increment increase in PM_{2.5} was associated with a 6% (95% CI: 4–8%) increase in all-cause mortality and 11% (95% CI: 5–16%) increase in CV mortality.40 The curve describing the dose–response relationship for the effects of air pollution on mortality appears to span from high levels of exposure to very low levels without evidence for a lower safe limit (Figure 2).

On the other hand, air pollution has also been linked with specific risk factors that may individually or collectively augment susceptibility to cardiovascular events over time. A recent systematic review and meta-analysis reported that most air pollutants (PM_{2.5}, NO₂, CO, and SO₂) with the exception of ozone are associated with a short-term increase (1–5%) in the risk of acute myocardial infarction (MI).42 Exposure to traffic has been linked to triggering of MI within 1 h and remained robust even after adjustment for multiple factors including prior physical exertion and time of the day.43 A sub-analysis of acute coronary events using 11 cohorts from the ESCAPE project reported substantial and statistically significant associations between PM_{2.5} and the incidence of acute coronary events, even at pollution levels below current European limits (12% increased risk per 10 μg/m³ in PM₁₀ and a 13% increased risk of coronary events per 5 μg/m³ increase in PM_{2.5} in > 100 000 participants from 11 cohorts across Europe).40 A meta-analysis involving 6.2 million events across 28 countries showed a small (≏ 1%) but significant association between admission for stroke/mortality and elevation in PM_{2.5} levels (/10 μg/m³) as well as gaseous pollutants (SO₂, NO₂, and CO) in the preceding 7 days.44 The risk of stroke increased by 17.5 and 19% (≏ 12 to 62%) per 5 μg/m³ increase in PM_{2.5} in the Women’s Health Initiative and ESCAPE studies in the USA and Europe, respectively.45,46 Chronic exposure to both PM₁₀ and NO₂ over a period of 12 years has also been shown to be associated with increased cerebrovascular mortality in China.47 Higher risk was especially seen in subjects above age 60 and in non-smokers and was consistently observed at PM concentrations <25 μg/m³. A positive association between short-term increases in gaseous components and PM_{2.5} and the risk of heart failure hospitalization or death has been shown.48 The effect estimates were stronger for the gaseous pollutants such as NO₂ and SO₂, with the former pollutant, suggesting that traffic-related air pollutants may drive this increase in risk. The association was more evident for patients with pre-existing CVD.48

Efforts over the last decade have focused on the links between air pollution and risk factors, with evidence to date indicating a link between exposure and enhancement of risk factors such as hypertension and type 2 diabetes mellitus. Numerous panel studies worldwide have also demonstrated a link between ambient levels of PM_{2.5}, black carbon, and other traffic-related pollutants with an elevation in blood pressure occurring within hours to days.49–51 In a large cohort of non-hypertensive individuals, every 10-μg/m³ increase of PM_{2.5} was associated with an odds ratio of 1.13 for the development of hypertension.52 The effect estimates for hypertension tended to be higher with traffic-related air pollutants, as shown by several studies on the association between black carbon, a marker of anthropogenic air pollution, and blood pressure (Supplementary material online, Table S3) as well as other measures such as NO₂ after adjusting for noise and concomitant medications (10-μg/m³ increase in NO₂ levels was associated with 1.34 mmHg (95% CI: 0.14, 2.55) higher systolic blood pressure).53 Controlled exposure studies have confirmed an effect of acute exposure to diesel exhaust and a rapid increase in blood pressure within a few hours.54 Similarly, air pollution has been associated with insulin resistance, diabetes, and obesity. In a meta-analysis of cohort studies involving a total of 2 371 907 participants and 21 095 incident cases of T2DM, the relative risk for diabetes increased by 39% per 10 μg/m³ of PM_{2.5}.55 A more recent meta-analysis that included 13 studies, PM_{2.5} and NO₂ increased risk of diabetes with HR of 1.10 (95% CI: 1.02, 1.18) and 1.08 (95% CI: 1.00, 1.17) per 10 μg/m³ increase in PM_{2.5} and NO₂, respectively.56 An increased incidence of insulin resistance and obesity in children, which indicate possible future development of diabetes, has also been shown. In two prospective cohorts, for every 2 SD increase in NO₂ and PM₁₀ (6 μg/m³), HOMA-IR increased by 17.0% (95% CI 5.0, 30.3) (10.6 μg/m³) and 18.7% (95% CI 2.9, 36.9), respectively.57 Similarly, multiple prospective studies have found an association between obesity in children and measures of traffic-related air pollution,58 but whether diabetes may represent a central mechanism for the increased long-term CV mortality associated with air pollution remains unclear.59

### Studies measuring impact of traffic noise and air pollution co-exposure on CVD

It is not common in air pollution to include measures of noise levels and conversely, most noise studies do not address the issues of...
concomitant co-exposure to air pollutants. A limited number of studies have measured both air pollution and traffic noise in the same study and have also performed adjustments in the overall statistical models (Supplementary material online, Table S1). Many of these studies appear to suggest that traffic noise and air pollution may independently contribute to the risk of cardiovascular incidence and mortality. A meta-analysis from 2013 found that correction for either air or traffic noise pollution did not change the association of the other with respect to CVD outcome.60 However, the association between road traffic noise and CVD disappeared after adjustment for air pollution (and vice versa) in some of the studies.51,62 This illustrates that the relatively high collinearity between road traffic noise and air pollution may complicate the interpretation of results of the mutual adjustment models. One problem is that often one exposure model predicts exposure levels more precisely than the other exposure model. If for example the air pollution model predicts air pollution more precisely than the noise model predicts road traffic noise, this would result in a more robust association between air pollution and outcome. Therefore, research in noise exposures relatively independent of air pollution, such as aircraft noise and railway noise, is a very important input in investigating whether traffic noise is an independent predictor of cardiovascular risk. Although there is a large body of evidence suggesting that air pollution exposure effects are not due to noise, research to evaluate confounding and combined effects is important, particularly regarding traffic air pollution effects remains important to consider.

Implications for global cardiometabolic health

Co-exposure to air and noise pollution is endemic to modern/urban societies and are growing public health issues faced throughout the world. While the burden of disease due to co-exposure is currently hard to surmise, given the uncertainties of the limited studies to date, it is clear that current estimates of disease burden due to each risk factor individually, even when using sophisticated approaches, such as those employed in the global burden of disease statement, are likely underestimates.63 By impulsively promoting the development of hypertension and diabetes, both environmental factors may pose a further and even greater public health burden. Unfortunately, countries with the highest rates of both cardio-metabolic disorders (e.g. India), also suffer the highest levels of PM$_{2.5}$ and noise exposures. Thus, the rapidly burgeoning epidemics of hypertension and type 2 diabetes in many economies in Asia may be much more than a simple coincidence of overlapping prevalence rates due to urbanization and adoption of western lifestyles. Hypertension affects 30–40% of the adult population worldwide (> 1 billion people) and disproportionately impacts developing nations (e.g. Africa, India, and Asia). In a similar manner, the prevalence of diabetes is burgeoning with the greatest increases rampant throughout the developing world. Based on the data to date, it is reasonable to speculate that environmental factors may also play a facilitatory role and may contribute to the collective burden of CVD worldwide through potentiation of intermediate risk factors.

Strategies to mitigate cardiovascular effects of noise and air pollution

While unfortunately no data on the effects of noise mitigation on CV outcomes are yet available, several natural experiments have provided an interesting opportunity to test the impact of reductions in air pollution on CV and mortality outcomes. These studies in aggregate demonstrate an independent improvement in life expectancy and a reduction in CV events after reduction of the exposure (Supplementary material online, Table S4). Over two decades (1980 through 2000), reductions in fine PM air pollution in the USA were associated with significant improvements in life expectancy, even after controlling for changes in socioeconomic, demographic, and smoking variables. Recently, a follow-up study found further increases in life expectancy, with additional reductions in air pollution between 2000 and 2007 (Supplementary material online, Table S4). Data from personalized intervention studies are also available, demonstrating that use of domestic air-filtration devices, particle-filtration masks, and car air filtration/air conditioning leads to meaningful reduction in CV surrogates such as systolic blood pressure, improvements in microvascular function, autonomic tone, and lower levels of inflammatory biomarkers in adults exposed to PM$_{2.5}$ (Supplementary material online, Table S5). The extent of these changes appears to be similar, if not larger, than that associated with interventions like salt reduction, such that correction of background air pollution should be seen as a priority for population-based preventive strategies.

However, efforts to mitigate air pollution and noise are a complex endeavour as they involve addressing their sources, which vary depending on the country and region of the world and complex economic and geopolitical considerations. Measures such as mandatory or voluntary greenhouse emissions or fuel efficiency standards, shifting to lower-carbon fuels, legislating the use of motorized vehicles/kilometres driven, introduction of electric mass transit, congestion pricing/taxes, vehicle and fuel taxes, and advanced vehicle technologies (e.g. battery electric cars, hybrids, plug-in hybrids, and fuel cell cars) may help simultaneously alleviate air/noise pollution and climate change goals. In addition, quieter tires and noise barriers are important tools in reducing road traffic noise. However, population growth, urbanization, and economic development imperatives are widely anticipated to offset any short-term gains. Even in a scenario of decreasing per-vehicle emissions, a global economy that relies heavily on the transport of goods over large distances, increasing industrial and transportation activity close to communities and poor land use planning may offset any gains. Urban planning that include land use assessment, requiring minimum distances between sources and individuals, incorporation of taller sound-wall barriers, relocation of traffic sources (including major trafficked roads but also airports) away from heavily crowded areas, avoidance of mixed-use areas (industrial—residential) and better roads are important in reducing co-exposure to air and noise pollution. Modification of the infiltration of outdoor pollutants and noise into indoor environments, which is largely a function of air exchange and building design, may offer further opportunities for exposure reduction to both noise and air pollution.
Evolutionary improvements in transportation technologies are likely to continue to reduce air pollution and noise, but may take time to implement, as many of these are expensive and may be hard to implement in countries, where cost is a primary consideration. In the meantime, there are opportunities to intervene at a personal level to reduce exposure. Increased awareness of the societal burden posed by these risk factors and acknowledgement in traditional risk factor guidelines may pressurize politicians to intensify the efforts required for effective legislation on the reduction of air pollution and noise.

Summary and conclusion

An effective reaction to the issues of air and traffic noise pollution requires a paradigm shift in human activity and will need large-scale macro intervention. From a regulatory standpoint, developing separate standards for environmental risk factors such as air and noise pollution is important. Although challenging to implement, combinatorial regulatory approaches for air and noise pollution, given the co-localization of these pollutants and the potential co-benefits of reducing both, a deviation from the current approach may need to be considered and approaches that investigate and tackle multiple environmental risk factors simultaneously, in the context of improving urban form leading to key co-benefits such reduced greenhouse gas emissions, greater resilience to climate change and increased physical activity of the population need to be considered, in order to maximize the health co-benefits of reducing their exposure. Given the growing body of evidence linking air pollution and noise pollution as significant modifiable risk factors, these stress factors may need to be considered in the pathogenesis of CVD and may be worthy of mention in current guidelines for cardiovascular prevention, NSTEMI, STEMI, and congestive heart failure.

Supplementary material

Supplementary material is available at European Heart Journal online.

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