Combined effects of air and noise pollution

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ABSTRACT

Air pollution exposure may be a confounder in epidemiological studies on health effects due to noise pollution, and vice versa. This paper summarizes recent existing evidence for myocardial infarction due to transportation noise and air pollution exposures; the risk of incident heart failure due to long-term exposure to road traffic noise and nitrogen dioxide; the risk of incident hypertension due to road traffic noise and air pollution exposure; the link between long-term exposure to transportation noise and air pollution exposure and incident diabetes; and the possible relation between exposure to noise and air pollution and dementia. Essential conclusions are: Transportation is associated with myocardial infarction independent of air pollution, but air pollution studies not adjusting for noise exposure may overestimate cardiovascular disease burden of air pollution. High exposure to road traffic noise and nitrogen dioxide was associated with the highest risk of incident heart failure, mainly among men No clear association appears to exist between incident hypertension, air pollution and road noise. Road and aircraft noise seem to be more relevant than railways noise and nitrogen dioxide in diabetes development.

INTRODUCTION

Common sources of air pollutant concentrations and sound pressure levels include road and motorway traffic, aircraft transport at starting, taxiing and landing operations, industrial compounds, power plants, commercial and hospitality establishments, electricity generators, and other stationary engines.

Health effects of exposure to noise include [1],[2]:

- Cardiovascular and other physiological effects,
- Cognitive impairment,
- Effects on sleep,
- Psychological effects including those on performance and memory,
- Annoyance.

Health effects from exposure of people to the "classical" air pollutants sulphur dioxide, nitrogen dioxide, ozone, carbon monoxide, and suspended particulate matter include [3],[4]:

- Cardiovascular morbidity and mortality,
- Effects on development,
- Effects on the central nervous system, and
- Odour annoyance.

With respect to cardiovascular effects noise and air pollution affect the same health endpoint as has been convincingly discussed by Münzel et al. in their analysis of both the pathophysiology and epidemiology of both noise and air pollution of cardiovascular disease [5]. Outdoor air pollutants

that can influence the cardiovascular system besides noise include particulate matter, carbon monoxide, ozone and sulphur dioxide. Figure 1 illustrates the potential pathways.

After a brief overview of the development relating to the consideration …. health impacts of both air pollution and noise In this paper we discuss the evidence of noise-outdoor-air-pollution interaction and corresponding adverse health impacts. The paper will not consider the interactions of noise exposure with indoor air pollution exposure; noise with asphyxiants; noise with solvents; noise and heavy metals; noise and pesticides; and noise and biological agents.

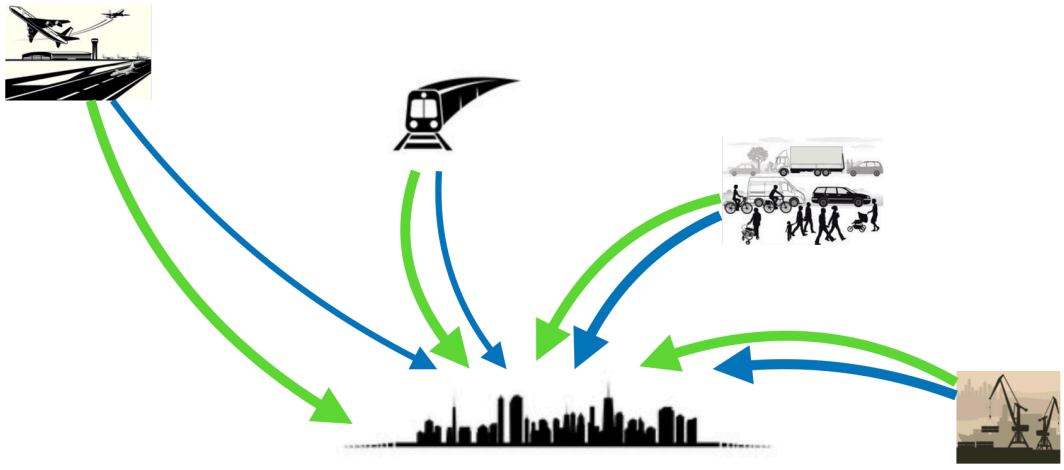

Figure 1: Potential pathways for induction of cardiovascular disease by transportation-related noise and air pollution

Green arrows: noise
Grey arrows: air
Source: Adapted from [6]

This article is based on an invited presentation given at the “Safe Sound Parliament Meeting”, Trivandrum, India, 24 - 25 August 2019 [7].

HISTORICAL BACKGROUND

Exposure to environmental noise and outdoor air pollution occur commonly, in particular with respect to road traffic. Early studies have supposed an interaction of exposures to environmental noise and outdoor air pollution with respect to annoyance due to noise or smell in different community environments,[8],[9],[10],[11],[12],[13],[14]. The conclusion from these studies was that exposure response relationships for noise and air pollution annoyance should include environmental context factors apart from noise and air pollution indicators [15],[16],[17],[18].

In a paper contributed to and a presentation given on behalf of WHO at the 12th IUAPPA Clean Air and Environment Congress by Schwela the possible interaction of noise and air pollutant indicators was discussed with respect to cardiovascular morbidity [19]. It was concluded that investigations on impacts of air pollution on cardiovascular health should consider noise exposure as a potential confounding variable. In a subsequent paper the authors insisted that most studies of health impacts of physical and chemical agents in the environment considered the health effect as being solely due to the air pollutant(s) under investigation, for example, air pollution without due regard for the simultaneous presence of noise pollution whereas both have an impact on the cardiovascular system; or noise without investigating the contribution of solvent, asphyxiant or metal exposures whereas they can have an impact on hearing impairment [20](Schwela et al., 2005). The influence of other factors, which can confound noise studies also need to be weighted with the existing evidence on the association of noise and air pollutant exposure and associated health impacts [18].

In 691 subjects in Augsburg, Germany, a strong association between onset of myocardial infarction (MI) and traffic exposure within the past hour was reported by Peters et al., although whether this was a result of the air pollution or a combination of other factors (eg, noise and stress) was not considered certain [21](Peters et al., 2004)

At the EC-JRC 2007 International Workshop on combined environmental exposure: noise, air pollutants and chemicals it was concluded that the health impacts of the combined exposure to noise, air pollutants, and chemicals are rarely considered in epidemiological studies [18]. Little is known if the pathogenesis of allergies can be stimulated particulate matter from diesel exhaust and NO_2 as well as noise; whether prevailing concentrations of air pollutants and chemicals can lead to ototoxic health impacts; and whether lead and noise combined can exacerbate hearing loss. As a result of the Group work during the Workshop [18] the risk of combined exposure to noise and specific air pollutants is illustrated in Table 1.

Table 1: Combination of noise with outdoor air pollutants [17]

Outdoor air pollutant	Exposure	Location	Risk
Particulate matter	Yes	Traffic areas; Industrial areas	High
Nitrogen dioxide	Yes	Traffic areas; Industrial areas	Medium
Sulphur dioxide	Likely	Industrial areas	Low
Carbon monoxide	Yes	Traffic areas	High
Mercury	Likely	Urban areas	Medium

As a consequence the Workshop participants recommended that highest priority should be given to detailed assessments of combined exposures to noise, vibrations and particulate matter (PM), carbon monoxide (CO), nitrogen oxides (NO_x), and volatile organic compounds (VOCs) with specific studies in urban areas and, especially, cardiovascular health endpoints [18].

Davies et al. reported a Pearson correlation above 0.5 between nitrogen oxides and an equivalent sound pressure level $L_{eq,5min}$ for 103 urban sites with varying traffic, environment and infrastructure characteristics. This observation lead to a recommendation to measure both pollutants in future studies of traffic-related pollution and cardiovascular disease (CVD) to allow for more sophisticated analysis of this relationship [22].

An analysis from a cohort study in the Netherlands demonstrated that several metrics of traffic-related air pollution exposure remained significantly associated with increased risk for cardiovascular events even after adjustment for higher levels of traffic noise [23]).

In a population-based case-control study on MI Selander et al. the correlation between long-term individual exposure to noise and air pollution from traffic was relatively high, R^2=0.36 [24]. However, the adjusted odds ratio for MI associated with long-term road traffic noise exposure was high, but did not indicate a strong influence of air pollution, lending some support to the hypothesis that long-term exposure to road traffic noise increases the risk for MI.

In 2010 the American Heart Association (AHA) reviewed the studies on the the causal relationship between traffic-related $PM_{2.5}$ and cardiovascular morbidity and mortality and noted that “despite the consistent epidemiological findings, these studies have yet to elucidate which of the many pollutants or other associated risks (i.e. noise) produced by traffic are responsible for the increase in risk for(CVD)” [25]. The AHA, therefore, concluded that epidemiological studies on CVD associated with air pollution could be improved by accounting for transportation noise.

A meta analysis by Fuks et al. of seven cohort studies of ESCAPE (European Study of Cohorts for Air Pollution Effects) considering exposure to road traffic noise and $PM_{2.5}$ (particulate matter less than 2.5 μm), soot, NO_2 (nitrogen dioxide) did not establish a clear association between incident measured hypertension, air pollution and road traffic noise [26].

A five year exposure study by Gan et al. on individuals aged 45-85 exposed in metropolitan Vancouver to community noise and traffic-related $PM_{2.5}$, NO_2 and nitric oxide (NO) observed that there are independent effects of traffic-related noise and air pollution on coronary heart disease (CHD) mortality [27].

A systematic review of studies by Tétreault et al. on the confounding effect of traffic-related noise or air pollutants on the association between the exposure to one or the other and cardiovascular effects seems to suggest that confounding of cardiovascular effects by noise or air pollutants is low [28]. Nine publications were identified. For most studies, the specified confounders produced changes in estimates smaller than10 per cent. The correlation between noise and pollutants, the quality of the study and of the exposure assessment do not seem to influence the confounding effects to properly assess if noise and air pollution are subjected to confounding. The authors admit, however, that improvements in exposure assessment are needed.

Floud, et al. used the data of the HYENA (Hypertension and Environmental Noise near Airports) study to assess potential confounding by NO_2 of the cross-sectional associations between self-reported 'heart disease and stroke' and aircraft noise and road traffic noise [29]. Data were analysed for three airports. The association found between night-time average aircraft noise and 'heart disease and stroke' was found after adjustment for socio-demographic confounders for participants who had lived in the same place for more than 20 years was robust to adjustment for exposure to air pollution. However, 24 hour average road traffic noise exposure was associated with 'heart disease and stroke', but adjustment for air pollution suggested this may have been due to confounding by air pollution.

In a randomized, crossover study by Huang et al., 40 young healthy adults were exposed for two hours to traffic-related $PM_{2.5}$, black carbon (BC), CO and noise at two different locations (traffic hotspot, park) and ambulatory electrocardiograms were taken [30]. Traffic-related air pollution and noise were both associated with alternations in heart rate variability (HRV). Effects of air pollutants were amplified at high noise levels(>65.6 dBA) as compared with low noise levels (<65.6 dBA). The study provided evidence that both traffic-related air pollutants and noise are associated with HRV, which may increase the risk for cardiovascular events.

In order to better understand the relative contribution of noise exposure and air pollution from transportation sources to health, in particular cardiovascular ailments, Stansfeld identified twenty five primary research studies between 2007 and 2015 that included assessments of noise and air pollution exposure from road, railway and aircraft and hypertension, atherosclerosis, ischaemic heart disease, stroke and mortality [31]. The quality of the studies was assessed in terms of population representativeness, modelling and/or measurement of noise and air pollution exposures, sufficient adjustment for confounding factors and measurement of health outcomes. From the review of these studies the author concludes that there is "good evidence of traffic noise effects on cardiovascular outcomes that are only minimally diminished, on the whole, by air pollution". However, it is recommended that policymakers and urban planners should take both exposures into account when regulating environmental impacts.

Within the longitudinal Scania Public Health Cohort survey in Skåne, Sweden, Bodin et al. studied a stratified random sample of persons aged 18–80 years with respect to the incidence of MI in 2000, 2005 and 2010 [32]. Both road traffic noise (L_{den}) and air pollution (NO_x) have been estimated simultaneously with a mean of 52 dBA and of 11 μg/m^3, respectively. After adjustment for individual confounders the MI incidence rate did not increase with a 10 dBA increase in L_{den} or with a 10 μg NO_2/m^3 increase.

A cohort of participants from the Myocardial Ischaemia National Audit Project in Greater London was followed by Tonne et al. from 2003-2010 for death or readmission to hospitals for MI [33|. Their residential exposure to air pollution (PM, NO_x, O_3) and traffic noise (L_{Aeq16}, distance from heavy goods vehicle traffic)) was modelled in urban and peri-urban environments. Covariates (age, gender, ethnicity, smoking and medical history, in-hospital treatment, medication) were included in estimating hazard ratios by use of Cox proportional hazards models. Covariate-adjusted hazard ratios for combined all-cause mortality and hospital readmission were above 1 for $PM_{2.5}$, PM_{10}

(particulate matter less than 10 μm), NO_2, and NO_x but not for ozone (O_3), within urban and peri urban areas. Adjusted for noise indicators these hazard ratios did not significantly change indicating a modest association with prognosis independent of air pollution.

In a postal survey study on the traffic annoyance rating at various areas in Auckland motorway, non-motorway, and central suburb) and Wellington (airport, non-airport, and rural) Shepherd et al. [34] focused on the covariance between air pollution annoyance and noise annoyances, and their independent and combined effects on the Health-Related Quality of Life (HRQOL). Air pollution annoyance and noise annoyance had the highest ratings for the residents living close to the motorway and the lowest ones were observed for residents in the rural area. For these two areas statistically significant differences existed between air pollution annoyance and noise-related annoyance, which were absent in the other areas. Bivariate and partial correlation coefficients (controlling for age, gender, education, illness and noise sensitivity) representing the relationship between air pollution annoyance and noise annoyance were significantly associated across all six areas. Mean scale scores for the overall quality of life (QOL), self-reported health, physical HRQOL, psychological HRQOL, social HRQOL, and environmental HRQOL decreased with annoyance ratings both for annoyance to air pollution and annoyance to noise exposures. Air pollution annoyance showed greater variability in the physical HRQOL domain while noise annoyance showed greater variability in the psychological, social and environmental domains. In consequence air pollution and noise appear to impact on health independently [34].

In conclusion, up to and including the year 2016, there is no clearly agreed evidence for the impacts of transportation noise **and** air pollution on cardiovascular diseases, hypertension, atherosclerosis, stroke and mortality.

In the next sections a brief overview of the more recent literature of the interaction of noise and air pollution relating to some health endpoints is presented.

CARDIOVASCULAR DISEASE DUE TO TRANSPORTATION NOISE AND AIR POLLUTION EXPOSURE

Analysing road traffic noise, air pollution and cardiovascular health data from three large cohort studies in England (HUNT, EPIC-Ocford. and UK Biobank), Cai et al. investigated the associations between long-term residential exposure to road traffic noise (L_{den}), outdoor air pollution ($PM_{2.5}$, PM_{10}, NO_2) and incident CVD, ischaemic heart disease (IHD) and cerebrovascular ailments (stroke), taking into account effects of potential confounders and co-adjustment of both road traffic noise and air pollution [35]. Results were presented as hazard ratios and their 95% confidence intervals per an interquartile range of noise/air pollution exposure. Significant positive hazard ratios for long-term PM_{10} or $PM_{2.5}$ exposure and all incident CVD were observed after adjustment for noise. No significant hazard ratios between road traffic noise and incident CVD were established in the sample of over 355,000 individuals. Evidence from this study suggested a possible association between road traffic noise and incident IHD.

MYOCARDIAL INFARCTION DUE TO TRANSPORTATION NOISE AND AIR POLLUTION EXPOSURE

The objective of a cohort study performed by Héritier et al. was to disentangle the risk for MI from exposure to transportation noise from road, railway and aircraft traffic and from air pollutants $PM_{2.5}$ and NO_2 [36]. Exposures to transportation noise and to air pollutants were modelled for the addresses of 4.4 million participants aged above 30 years of the Swiss National Cohort (SNC) study during a follow-up period 2000-2008. Estimated noise indicators were L_{den} (road), L_{den} (railways), and L_{den} (aircraft). Daily $PM_{2.5}$ at 100m grid cells across Switzerland was estimated for 2003–2008 from satellite, land use, meteorological data, and $PM_{2.5}$ ground measurements. NO_2 exposures were estimated from ground monitoring stations of 11 cantons and the ‘Ostluft’ (Air surveillance in the eastern cantons) and ‘Inluft’ (Air surveillance in central Switzerland) networks of the SAPALDIA (Study on Air Pollution And Lung Disease In Adults) Cohort. Data on MI mortality were taken from the SNC database. Data were analysed using the Cox proportional hazards model for participants living in Switzerland during 2003-2008. Confounders included age, gender, socio-

economic position, civil status, educational level, nationality, and mother tongue . MI risks associated with noise were estimated through hazard ratios HR_n for 10 dB increase in traffic noise, see table 2. MI risks associated with air pollutants were estimated through hazard ratios HR_a for 10 µg/m^3 increase in pollutants, see Table 3.

These tables show that MI mortality due to transportation is independent of air pollution because H_a decreases when adjusted for noise and H_n remains unchanged when adjusted for air pollution. Air pollution studies not adjusting for noise exposure may, therefore, overestimate MI mortality and consequently the cardiovascular disease burden of air pollution.

Table 2: Hazard ratios for MI mortality due to air pollution increment adjusted for transportation noise

Air pollutant	HR_a not adjusted (95% CI)	HR_a adjusted for noise (95% CI)
$PM_{2.5}$ (10 µg/m^3)	1.052 (1.013-1.093)	1.019 (0.971-1.071)
NO_2 (10 µg/m^3)	1.024 (1.005-1.043)	0.990 (0.965-1.016)

Table 3: Hazard ratios for MI mortality due to noise increment adjusted for air pollution

Noise	HR_n not adjusted (95% CI)	HR_n adjusted for $PM_{2.5}$ & NO_2 (95% CI)
Road traffic (10 dB)	1.032 (1.014-1.051)	1.034 (1.014-1.055)
Railways (10 dB)	1.020 (1.007-1.033)	1.020 (1.007-1.033)
Aircraft (10 dB)	1.025 (1.006-1.045)	1.025 (1.005-1.046)

LONG-TERM EXPOSURE TO ROAD TRAFFIC NOISE AND NO_2 AND RISK OF INCIDENT HEART FAILURE

In a cohort study on the risk of incident heart failure in first-ever hospital admissions of over 57,000 people aged 50-64 years Sørensen et al. aimed to investigate the association of this health endpoint with road traffic noise and NO_2 during an exposure period of 13 years [37]. Level of road traffic noise (L_{den}) were estimated at all participants' addresses by a calculation programme that implements the joint Nordic prediction method for road traffic noise. Exposure to annual levels of NO_2 for all addresses were estimated by a dispersion model. Exposures for both indicators were modelled as time-weighted means 1-, 5-, and 10-y preceding diagnosis , taking all then current and historical addresses in these periods into account. Analyses were based on Cox proportional hazards model by adjusting incidence rate ratios (IRR) for heart failure to mutual exposure to noise and NO_2. Confounders included sequentially age, gender (Model 1), lifestyle, socio-economic status, calendar year (Model 2) and NO_2 and noise (Model 3). Figure 2 shows the IRR for the risk of incident heart failure due to 10-year exposure to road traffic noise and NO_2.

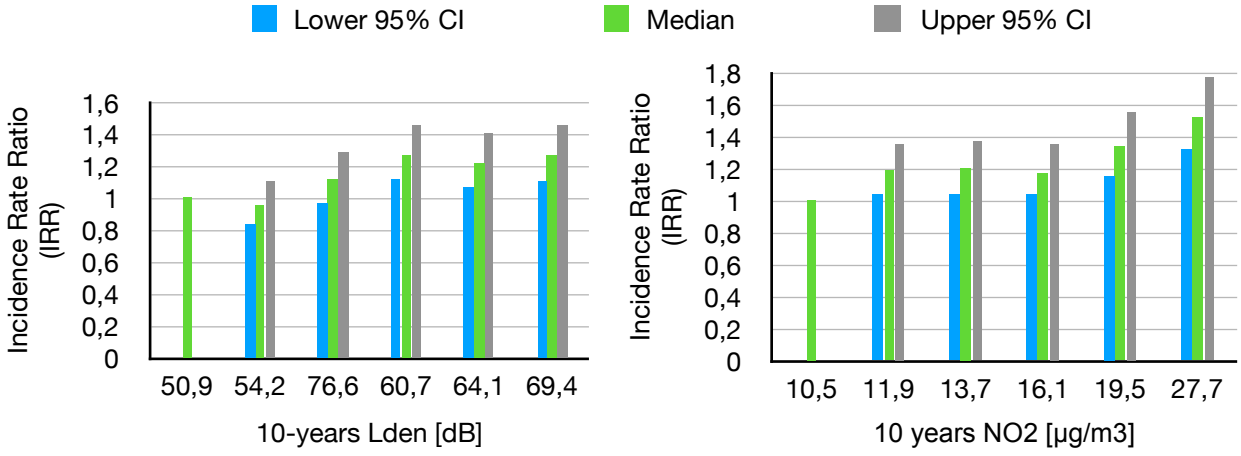

Figure 2: Incidence Rate Ratios for incident heart failure due to 10-year exposure to L_{den} and NO_2

From Figure 2 it is to be concluded that increasing exposure to road traffic noise and NO_2 is associated with increasing risk of incident heart failure.

Table 4 shows the IRR of the three models per interquartile range of exposure.

Table 4: IRR and 95% CI for exposure to road traffic noise and NO2

Exposure (per interquartile range)	IRR (Model 1)	IRR (Model 2)	IRR (Model 3)
L_{den} (9.9 dB)	1.24 (1.18-1.31)	1.14 (1.08-1.21)	1.08 (1.00-1.16)
NO_2 (7.5 µg/m^3)	1.20 (1.15-1.25)	1.11 (1.07-1.16)	1.07 (1.01-1.14)

Model 1: Adjusted for age and sex
Model 2: Model 1 + adjusted for lifestyle, socio-economic status, calendar year
Model 3: Model 2 + adjusted mutually for NO_2/L_{den}

According to Table 4 incident heart failure is strongly associated with exposure to road traffic noise and NO_2.

INCIDENT HYPERTENSION DUE TO ROAD TRAFFIC NOISE AND AIR POLLUTION EXPOSURE

In a cross-sectional study of cardio-metabolic diseases (heart attack, stroke, diabetes and hypertension), Klompmaker et al. [38] investigated the associations between these diseases among more than 354,000 adults with with surrounding green area indicators (normalized difference vegetation index- NDVI 300m), modelled annual average air pollution indicators (NO_2 ,$PM_{2.5}$, oxidative potential with dithiothreitol - OP^{DTT}, among others) and modelled road-traffic noise indicators (L_{den}). The associations were analysed by logistic regression via estimation of odds ratios (ORs) and their 95% confidence intervals (CIs) in single, and joint odds ratios (JORs) for surrounding green area, air pollution and traffic noise indicators and two-, three- and four-exposure regression models.The JORs are based on the Cumulative Risk Index (CRI) method of combined exposure to air pollution, traffic noise and surrounding green. They are defined as the odds of a one-unit change of IQR increase in air pollution and traffic noise indicators and a corresponding decrease in surrounding green areas relative to the odds of no change under the assumption that the effects of air pollution and noise exposure and green areas were additive.

Based on the findings of the single-exposure models, the authors decided to focus on hypertension and diabetes (see next section)) in multi-exposure models. Table 5 shows the results for of single regression models of ORs for two of the cardio metabolic diseases

Table 5: Single-exposure regression odds ratios for hypertension and diabetes

Exposure variable	Hypertension	Diabetes
NDVI 300m	0.97 (0.96,0.99)	0.91 (0.89,0.93)
OPDTT	1.02 (1.00,1.03)	1.06 (1.04,1.09)
NO2	1.02 (1.00,1.03)	1.06 (1.04,1.09)
PM2.5	1.01 (1.00,1.02)	1.01 (0.99,1.03)
Lden	1.00 (0.99,1.01)	1.02 (1.00,1.03)

Four-predictor regression analysis for hypertension for the JOR of NDVI 300m + OP^{DTT} + NO_2 + L_{den} did not change the three-predictor regression for the JOR of NDVI 300m + OP^{DTT} + NO_2, indicating that air pollution as estimated by OP^{DTT} and surrounding green as estimated by NDVI, but not road traffic noise, were associated with hypertension.

LONG-TERM EXPOSURE TO TRANSPORTATION NOISE AND AIR POLLUTION EXPOSURE AND INCIDENT DIABETES

Based on the SAPALDIA cohort Eze et al. [39]aimed to investigate the independent effects of noise (road, aircraft and railway noise and specific noise characteristics like number and temporal

variation of noise events), and of NO_2 on diabetes incidence in adults. Incident diabetes was identified in a sample of over 2,600 participants who were not diabetic at the beginning of the study. Participants answered questions regarding their health and medication use and the diabetes indicator glycated haemoglobin was measured. Road traffic, railway and aircraft sound pressure levels were estimated as annual means of the indicators $L_{eq,day}$ (7-23 h; dB), $L_{eq,night}$ (23-7 h; dB) and L_{den} (dB) for road, railway and aircraft noise using various Swiss noise models. NO_2 exposure was estimated through a Gaussian dispersion model at the participants' residence addresses. Confounding variables included age; gender, education; socio-economic index; smoking behaviour; passive smoking; alcohol consumption; consumption behaviour with respect to fruits and vegetables; physical activity; and body mass index. The relative risk (RR) of diabetes and its 95% CI relating to transportation noise and NO_2 exposures was estimated by Poisson and logistic regression models, respectively. Figure 3 show the RR for incident diabetes relating to transportation noise (L_{den}) and NO_2 from multi exposure models including all confounding variables. Similar plots for $L_{eq,day}$ and $L_{eq,night}$) show very similar relationships due to their high correlation to L_{den}.

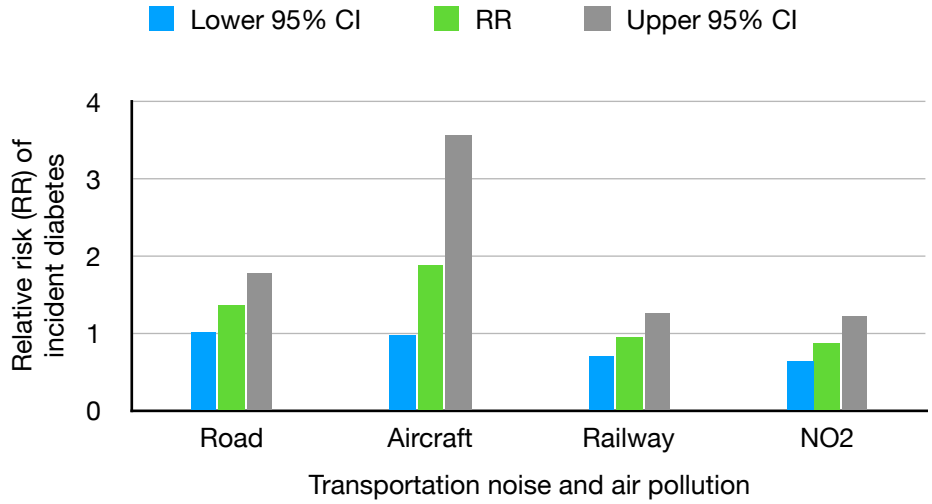

Figure 3: Relative risk of incident diabetes related to interquartile range of L_{den} (road, 10 dB), L_{den} (aircraft, 11 dB), L_{den} (railway, 12 dB) and NO_2 (1.5 µg/m3)

RR(road) and RR(aircraft) for incident diabetes are significant and almost significant, respectively. Both L_{den}(railways) and NO_2 are not correlated with incident diabetes risk, in contrast to previous observation of a positive association between NO_2 and prevalent diabetes in previous studies of Eze et al., a fact that requires more investigation on the relationship between incident diabetes and transportation noise and air pollution [40],[41].

In the study of Klompmaker et al. mentioned in the previous section [38] the authors equally investigated the associations between diabetes and the normalized difference vegetation index NDVI (the proportion of green area within a buffer around a participant's residential address), NO_2, OP^{DTT} and L_{den}. The surrounding green area indicators were inversely associated with diabetes, while air pollutants (NO_2, OP^{DTT}) and road traffic noise were positively associated with diabetes, cp. Table 5. In two-exposure analyses, associations with green area and air pollution indicators remained, but the association between L_{den} and diabetes disappeared when adjusted for surrounding green or air pollution. Studies including only one of the correlated exposures of surrounding green, air pollution, and road traffic noise may overestimate the association of diabetes and hypertension attributed to the studied exposure. Visa versa, the joint impact of exposure to a combination of surrounding green and air pollution may be underestimated by the associations from single-exposure models.

COMBINED EXPOSURES TO GREEN AREAS, AIR POLLUTION AND TRAFFIC NOISE ON MENTAL HEALTH

In another cross-sectional study based on the same sample of participants, Klompmaker et al. [42] investigated the associations between poor mental health indicators (self-reported psychological distress, prescriptions of anxiolytics, hypnotics & sedatives, and antidepressants) with surrounding green area indicators ((NDVI), modelled annual average air pollution indicators (PM_{10}, $PM_{2.5}$, NO_2) and modelled road- and rail-traffic noise indicators (L_{den}). The associations were analysed by logistic regression via estimation of odds ratios and their 95% confidence intervals in single, and JORs for surrounding green area, air pollution and traffic noise indicators in two- and three-exposure regression models.

In single exposure models, the surrounding green area indicators were inversely associated with all four poor mental health indicators. All air pollution indicators were positively associated with all poor mental health indicators. Road-traffic noise was only positively associated with prescription of anxiolytics, while rail-traffic noise was only positively associated with psychological distress. In multi-exposure analyses, JORs were higher than the ORs of single exposure models. The authors conclude that studies including only one of these three correlated exposures may overestimate the influence of poor mental health, while underestimating the influence of combined environmental exposures. Thus, further research into the effect of air pollution and noise on mental health is needed as has been emphasised also by other authors [43],[44] (Ali & Khoja, 2019; Hegewald et al., 2020).

RELATION OF NOISE AND AIR POLLUTION TO DEMENTIA?

In a retrospective cohort study in London Carey et al. aimed to investigate whether incident dementia (Alzheimer's disease, vascular dementia, non-specific dementia) is related to air and noise pollution [45]. Among 75 practices in Greater London over 130,9000 participants during 2005-2013 were eligible for the analyses. Air pollution indicators were $PM_{2.5}$, $PM_{2.5}$ (traffic), NO_2, and ozone (O_3), while the main noise indicator was L_{night}. Confounding variables included age, gender, ethnicity, smoking, body mass index (BMI), the Index of Multiple Deprivation (IMD) used in England as a measure of poverty, and co-morbidities that were independently predictive of dementia (IHD, stroke, heart failure, diabetes). A Cox proportional hazards model with adjustment for confounders was employed to estimate hazard ratios (HRs) for dementia associated with air pollution and night-time noise (Table 6).

Table 6: Adjusted HRs for incident dementia by air pollutants and night-time noise

Exposure	IQR change [µg/m³] or [dB]	HR_3 (95% CI)	HR_4 (95% CI)
NO_2	7.47	1.16 (1.05-1.27)	1.15 (1.04-1.28)
$PM_{2.5}$	0.95	1.06 (1.02-1.12)	1.06 (1.01-1.13)
$PM_{2.5}$ (traffic)	0.58	1.08 (1.01-1.16	1.08 (0.99-1.18)
O_3	5.6	0.85 (0.76-0.94)	0.85 (0.76-0.96)
L_{night}	2.7	1.02 (0.97-1.08)	1.01 (0.98-1.03)

HR_3: Cox model adjusted for age, sex, ethnicity, smoking, BMI, IMD, and co-morbidities.
HR_4: Cox model adjusted for age, sex, ethnicity, smoking, BMI, IMD, NO_2, $PM_{2.5}$, and L_{night}

As indicated in Table 6 dementia is associated with NO_2 and $PM_{2.5}$ and weakly with L_{night}.

In their discussion of the strengths and weaknesses of the study the authors mention their concerns around the variability of dementia diagnoses in UK primary care and that dementia diagnoses on primary care databases may not be an accurate reflection of the true prevalence.

CONCLUSIONS

The recent studies presented here have indicated exposure to both air pollution ($PM_{2.5}$, NO_2) and environmental noise exposure as important risk factors for the health endpoints:

- ➢ Myocardial infarction (mortality)
- ➢ Incident heart failure
- ➢ Hypertension
- ➢ Diabetes
- ➢ Mental health

This observation agrees with the conclusions of Sørensen and Pershagen [6] in their discussion of the paper of Héritier et al. [36] on myocardial infarction due to noise and air pollution. Consequently, air pollution studies for these and possibly other health endpoints, e.g. children's cognition, newborn parameters, atherosclerosis, multiple sclerosis, stroke should adjust for noise exposure and vice versa. If not, their use for guideline setting, control options, air quality management and environmental noise management is limited.

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