Non-auditory effects of noise: an overview of the state of the science of the 2017-2020 period

Kerstin Persson Waye[1], Elise van Kempen[2],

[1] The School of Public Health and Community Medicine, Sound Environment and Health, Institution of Medicine, University of Gothenburg, Sweden.

[2] RIVM, Centre for Sustainability, Environment and Health Bilthoven, The Netherlands

Corresponding author's e-mail address: kerstin.persson.waye@amm.gu.se

ABSTRACT

This paper provides an overview of non-auditory effects of noise published since the last ICBEN conference in 2017. The paper focuses on the impact of environmental and occupational noise on cardiovascular and metabolic effects, mental health, dementia, birth outcomes inclusive congenital anomalies. The paper takes as a point of departure current reviews and adds new research from literature searches for peer reviewed journal articles in the large databases. The aim is to describe recent research achievements, emerging areas of research, remaining gaps of knowledge, and priority areas of future research in the field of non-auditory health effects of noise.

INTRODUCTION

Team 3 focuses on the non-auditory effects of occupational and environmental noise on long-term health outcomes including cardiovascular, physiological and endocrinological, mental health, birth outcomes and early child development and hospital noise effects on staff performance and patient rehabilitation. In October 2018, the Environmental Noise Guidelines for the European Region was published by WHO [1]. At the base of the Guidelines, were eight systematic reviews carried out detailing the strength of evidence for a number of outcomes for the noise sources: aircraft, road traffic, rail traffic and wind turbines. Most of the reviews cover a time frame between 2000 and 2014. The reviews were carried out according to a protocol that was specially developed for this purpose. As part of the review, the quality of the evidence that has been retrieved in an evidence review had to be assessed. To this end, the Grading Recommendations Assessment, Development and Evaluation (GRADE) approach [2, 3] was applied, a systematic and explicit approach making judgements about the quality of evidence. For every outcome, the quality of evidence was assessed according to several elements (e.g. study design, study quality, consistency of the results, directness of evidence. For WHO, the outcome of the assessment of the quality of evidence was of

importance, since the level of the quality of the evidence was by the so-called Guideline Development Group (GDG) linked with the guideline values and recommendations in the Guidelines.

For Team 3, the reviews on cardiovascular and metabolic effects (2), birth and reproductive outcomes (3) and mental health and wellbeing and quality of life (4) are most relevant. They form the starting point of our overview on the ICBEN-period 2017-2020. Due to the Covid-19 pandemic, the last ICBEN-period was prolonged with one year, to June 2021. The studies that have published during this extra year will also be taken into account.

Objectives

The aim for this overview paper is to describe recent research achievements, emerging areas of research, remaining gaps of knowledge, and priority areas of future research in the field of non-auditory health effects of noise. To this end, we have reviewed the literature on noise and health that has been published in the period 2017-2021. For congenital anomalies the period 2017-2020 was reviewed.

We have chosen to focus on cardiovascular and metabolic outcomes, birth outcomes inclusive congenital anomalies, mental health and dementia as well as describing emerging areas and outcomes to noise in general including both environmental and occupational sources.

METHODS

Our overview contained several steps: firstly all studies were identified that could potentially be included in the review. The identified studies were submitted to selection criteria. Studies that met these criteria were included for data-extraction. Finally, studies were summarized and compared. The following risk estimates are used and given in their abbreviated forms throughout the paper: Hazard Ratio (HR), Odds Ratio (OR) Relative Risk (RR), and presented with 95 percentage confidence intervals (95% CI)

Identification

We identified literature published between January 2017 and January 2021 about the impact of noise exposure on: (i) mental health and dementia (ii) birth outcomes including congenital anomalies, and for (iii) the cardiovascular system, (iv) and the metabolic system noise sources were specified as transport and wind turbines. Search terms were identified and discussed by the authors and followed largely the protocols of the WHO evidence reviews on noise and mental health, birth outcomes inclusive congenital anomalies, and cardiovascular- and metabolic effects as much as possible. In addition to the WHO-evidence review, we also tried to identify studies investigating the effects of occupational noise exposure in relation to birth outcomes including congenital anomalies. To this end, we adapted the search profiles applied in the WHO-evidence reviews for this purpose. The bibliographic databases that were utilized included PubMed, Scopus, Web of Science for the birth outcomes and congenital anomalies, PubMed and PsycInfo for mental health and dementia and Scopus MEDLINE, EMBASE, and PsycInfo for the cardiovascular and metabolic system. Within each database we screened all English papers in their respective period.

Selection

The publications that were identified by means of the search were selected for data extraction. To this end we applied the criteria that were developed in the three relevant WHO evidence reviews [4-6]. In summary, these criteria were: (a) Studies should report on the relationship between noise and exposure or (road, rail or air traffic noise exposure or wind turbine noise exposure), and cardiovascular and/or metabolic disease and/or mental health, or dementia and/or birth outcomes, in populations who were not identified with a certain illness or disorder; (b) With regard to environmental noise exposure, studies must quantify and/or describe the relationship between objective exposure (expressed in equivalent sound levels in decibels) and one or more of the relevant health outcomes. With regard to occupational noise exposure, we also allowed other measures of noise exposure.

In addition to the WHO evidence reviews, we also selected systematic reviews and meta-analyses that addressed that association between noise exposure and one or more of the outcomes relevant for the current review. For mental health we adopted largely the criteria developed in Clark and Paunovic [4] and added Dementia (vascular and Alzheimer) as well as hyperactivity to also capture ADHD disorders. As in Clark and Paunovic [4], we acknowledge that the term mental health refers to a number of mental health symptoms and often viewed as on a continuum with diagnoses that can be indicative of mental health disorders with various severity. For birth outcomes the criteria in Nieuwenhuijsen, Ristovska [6] were largely followed, though health outcomes related to the mother's pregnancy were not included. With regard to the cardiovascular and metabolic effects, we extended our selection criteria by also including studies that investigated the association between noise from road, rail, -and air traffic and wind turbines and (i) blood pressure in adults, (ii) additional cardiovascular diseases (including arterial fibrillation, arrythmia, heart failure, arterial stiffness, and BP-related death), and (iii) metabolic and cardiovascular risk indicators (including hsCRP, cholesterol, triglycerides, blood glucose, glycated heamoglobin, cortisol in saliva, glomerular filtration rate)

Data-extraction

From the studies that met the selection-criteria, we extracted the following data: (i) data on general study characteristics; (ii) population characteristics; (iii) exposure assessment; (iv) health outcome assessment, and (v) the results of the study. Due to the restricted time, we were not able to express the results in the same way, e.g. a Relative Risk per 10 dB change in noise level or the change in waist circumference per 10 dB change in noise. This also means that data will not be aggregated as part of a meta-analysis. In contrast to the WHO-evidence reviews we will also not assess the risk of bias per study. Instead, we will look at the quality of the studies in general.

We realize that a large amount of the overall certainty in the evidence comes from cross-sectional and ecological studies. However, in environmental health, cohort and case-control studies are often one of the highest quality evidence available to understand whether there is an association between an exposure and a health outcome. Due to limited space, we decided to mainly discuss the findings of the cohorts and case control studies. With regard to cardiovascular and metabolic outcomes, we additionally decided to present and discuss the findings of studies on hypertension, coronary heart disease (CHD), stroke, and diabetes only. For those who are interested the references of all identified studies can be found in reference list.

RESULTS

Results of the search- and selection process

Table 1 presents a flow of the sum of the search and selection process for the individual outcome groups, for the period 2017-01-01 – 2021-1-1.

Table 1: Sum of the search and selection process for the individual outcome groups

	Outcome group			
Phase in the process	**Mental health and dementia**	**Congenital anomalies**	**Birth outomes**	**Cardiovascular and metabolic outcomes**
Identification	386	578	91	893
Additional records identified through other sources	5	7	3	146
Records after duplicates removed	388	585	88	723
Records excluded	313	577	65	552
Full text articles assessed for eligibility	75	8	23	171
Full text articles excluded with reasons	34	2	7	86
Studies included for data-evaluation	13 reviews, 28 original studies	2 reviews, 4 original papers	3 reviews, 13 original studies	19 reviews, 44 studies (85 references)

Reviews

Mental health and dementia

For mental health we initially identified 28 reviews. Of those 13 are included in Table 2, while 10 were not deemed as systematic reviews, 4 not mental health-related, and 1 did not focus on noise. The majority of the reviews included transportation noises (road, air, rail), but there were single reviews of school yard noise, neighborhood noise, and occupational noise, and a couple of reviews of wind turbine noise and indoor noise. The majority focused on adult populations; only three reviewed the child population. Several of the reviewers and all meta-analyses were able to provide a quality judgement of the individual studies.

Meta-analyses were performed by two reviews and the most recent [7] found that depression risk increased by 12% with 10dB Lden (95%CI 1.02, 1.23) for aircraft noise. Road and rail noise indicated a small (2-3%) but not statistically significant risk increase. Dzhambov and Lercher [8] reported a tendency to 4% increase of depression and 12% increase of anxiety for road traffic noise. This review were on the basis of six studies able to provide a linear exposure response relation between depression and Lden ranging from 40 dB to 76dB, with a statistical significant increase found from about 55 dB. Compared to the findings of the WHO review [4] the above review included more longitudinal studies and point to some, albeit weak, association with mental health and aircraft and road traffic noise. Still the number of studies, especially for rail noise, was very low, and a better methodological quality should be strived for both with regard to outcome and exposure assessment.

Birth and reproductive outcomes, including congenital anomalies

Four reviews were included [6, 9-11] and only one of these performed a meta-analysis [10]. All these reviews focused on environmental sources of noise, mostly in the residential area, and evaluated exposure during pregnancy and outcomes in the offspring (Table 3).

Reviews point to the scarcity of publications regarding these outcomes, with a marked increase in publications recently. They suggest a small increase in the risk of adverse outcomes in association with environmental noise exposure. The reviewers were able to provide a quality judgement to the individual studies using different instruments. Overall, the quality of evidence for small for gestational age, pre-term birth and congenital anomalies was judged to be moderate to very low or low [6, 10]. For road traffic noise and low birth weight, there is some disagreement between the reviews whether the evidence is of low quality [6, 10] or whether there is already high quality evidence supporting no effect of road traffic noise on birthweight [9], based on the findings of two longitudinal studies. The meta-analysis suggest that a 10 dB increase in Lden was (marginally) associated with −8.26 g lower birth weight based on seven estimates [10].

Cardiovascular and metabolic outcomes

For our overview we included 19 systematic reviews [5, 11-28]. Their characteristics are presented in Table 4. Most of the studies that were covered by these reviews reported on the impacts of road and air traffic noise exposure. Eight reviews also included studies that investigated the impacts of rail traffic noise exposure [5, 13-15, 20, 23, 26, 29] and four reviews included studies that investigated the impact of wind turbine noise [5, 12, 23, 28]. Other sources under investigation were occupational noise (five studies) [12, 13, 18, 19, 27], recreational noise (one study) [27] and noise from humans (one study) [13]. Most reviews focused on adult populations; only four reviews included effects in children [5, 16, 23, 26]. The time range of the participating studies in the reviews was from 1947 to 2020. Ten reviews included one or more meta-analyses, resulting in more than 32 exposure-response estimates, usually in the form of a Relative Risk (RR), Odds Ratio (OR) or Hazard Ratio (HR) per 10 dB change [5, 13, 14, 16-19, 21, 25, 27]. For several reviews, the reviewers were able to provide a quality judgement of the individual studies. Several tools were applied such as IGN/CASP system, or the Newcastle Ottawa Scale (NOS) of the Agency for Health Research and Quality. In some cases also specially developed tools were applied [12, 16, 17, 28].This is an improvement in comparison with what was observed earlier in [5] where hardly any of the evaluated reviews were able to provide a quality judgement of the individual studies included in their review.

Table 2: Overview of identified systematic reviews of Mental Health and Dementia

First author and reference	Studies included								Meta- Analyses included
	Number evaluated	Number of participant	Time range	Countries*	Population	Noise source(s)†	Setting(s)	Health end point(s)‡	
Sakhvidi F Z., 2018[30, 31]	12	399 – 46,940, total 58,458	2001-2017	1 - 10	Children	A, B, C, D,	School, residence	CP, ES, SA, HA, I, SAtt; PB, OB, PR, A, MH	No
Clark C., 2018	29	Not specified	Jan 2005- Oct 15	10****	Adults, children	A, B, C	School, residence	D, A, HA, ES, CD, ED	No
Clark, C., 2019[9]	22	Not specified	MH 2015-19 Dement2014-19	1, 3, 4, 5, 7, 9 13, 14, 15, 21, 23	General, subgroups	A, B, C	Residence, school, classroom, public	I, D, A, MH ADHD, Anx, AntD, DM, PsyS, Dem, PD, Cdis;,	No
Dzhambov A M., 2019[8]	10	1477– 354,827	2015 – Aug -19	4, 5, 9, 11 - 15	Adults	B	Residence	AntD, Anx, D, A, DM	Yes
Freiberg A., 2019[28]	84(68 studies)	15 - 1277	2000 – 17	1, 2, 4, 5, 9, 11 – 13, 15 – 19, 21 - 25	All ages	E	Residence	PsyD, St, A, D, PsyS	No
Robbins R N., 2019[32]	10	289 – 4.4 million	2017 – Sept 18	3, 5, 15, 19, 24, 26, 27	Adults	Not specified	Urban and rural settings	Cdis	No

Schubert M., 2019[33]	14 (10 studies)	275– 46,940	Up to Feb -19	1 – 5, 9, 19, 21, 28	Children	A, B, C	School, residence	D, A, HA,I CD, ED, PR, PsyB	Yes
Dickerson, A., et al. 2020[34]	112 (13 incl. noise	144to 77,295 people	1976 - 2018	4, 5, 28, 12, 1, 14, 22, 21, 31, 32	Adults	A, B, H, I, F	School, residence, work	D, A, Su	No
Mucci, N., et al. 2020[35]	54 articles	32 to 971839 people	2010-2020	4, 12, 8, 10, 22, 19, 1, 2, 13, 31	Adults	A, B, C, E	Residence, work	PsyH	No
Peris, E et al. 2020[36]	76 articles	Not specified	2000-2017	****	Adults	A, B, C	Residence, natural	MH	No
Hegewald, J., 2020[7]	31	Not specified (5 386 270)	Up to Dec -19	1, 3, 4, 5, 9, 11, 12, 13, 14, 15, 22, 34	Adults	A, B, C, M	Residence	D, A, C dis, Dem	Yes
Huang, L., 2021[37]	59 (9 MH)	Not specified (3 937 708)	Up to Oct 20	3, 4, 5, 13, 15, 19	Adults	B, I, J, K, L	Resisence, work, urban	Dem, Cdis, Emerg hosp admiss. to D	No
Lan, Y., 2020[38]	11	Not specified (385 557)	Up to Feb 20	3, 4, 5, 9, 11, 12, 13, 15, 24, 34	Adults, or all ages	A, B, C, M	Residence, urban, neighborhood	A	Yes

*: 1 = Norway, 2 = Denmark, 3 = Spain, 4 = Germany, 5 = United Kingdom, 6 = Macedonia, 7 = Bulgaria, 8 = Austria, 9 = Netherlands, 10 = EU, 11 = Greece, 12 = Italy, 13 = Sweden, 14 = Finland, 15 = Canada, 16 = Australia, 17 = New Zealand, 18 = Poland, 19 = United States, 20 = Portugal, 21 = South Korea, 22 = Japan, 23 = Belgium, 24 = China, 25 = Switzerland, 26 = Ireland, 27 = Mexico, 28 = Serbia, 29 = Taiwan, 30 = South America, 31 = Global, 32= India, 33 = Iran, 34 = France

: Various states in South America, *: 60 countries, ****: Not further specified

†: A = Aircraft, B = Road traffic, C = Rail traffic, D = Schoolyard, E = Wind turbine, F = E-Noise G = Industry and Machine, H = indoor noise, I = Neighborhood noise, J = Occupational noise, K = Unspecified traffic noise, L = Road proximity, M = Combined sources of traffic

‡ CD- Conduct disorders; ED= Emotional disorders, SA= Social adaptability; HA=Hyperactivity, I=Inattention; SAtt= Sustained attention, PB=Prosocial behavior; OB= Opposing behavior, PR=Peer relationsship, ADHD = Attention Deficit Hyperactivity Disorder, A=Anxiety, AntD=Antidepressant, Anx= Anxiolytics, Cdis=Cognitive Disorders D=Depression, Dem= Dementia, DM= Depressed mood, MH=Mental health, PsyB= Psychosocial behavior, PsyD=Psychological distress, PsyH=Psychological health, PsyS Psychosocial symptoms, PD = Parkinson's disease, Stress= St, Su=Suicide

Table 3: Overview of identified systematic reviews of Birth outcomes, including Congenital anomalies

First author and reference	Studies included								Meta-analysis included
	Number evaluate	Number of participants	Time range	Countries*	Population	Noise source(s)†	Setting(s)	Health end point(s)‡	
Rugel and Brauer 2020[11]	51 (10 on noise)	1506 – 540,365	Up to Nov 2019	1, 2, 4, 5, 6,8 9, 10	Mothers or newborns	B, E, G	Residence	A, B, C, D, I, J	No
Clark, C., 2019[9]	7	518 – 540,365	January 2017 to March 19	4, 6, 11, 12	Live births, pregnant women	B, C, D	Residence	A, B, C, D, F,	No
Dzhambov A M., 2019[10]	9	518 – 540,365	2016 – May 19,	1 - 12	Live births, still births	B, G	Residence	A, B, C, D	Yes
Nieuwenhuijsen M J., 2017[6]	14	200 – 298,705	June 2014 – Dec 16	1, 2, 4, 13 - 16	Births (not furth. specified)	A, B, F	Not specified	A, B, C, D, E, F, G, H, I, J	No

*: 1 = Canada, 2 = Spain, 3 = Germany, 4 = Denmark, 5 = France, 6 = United Kingdom, 7 = Sweden, 8 = Lithuania, 9 = Norway, 10 = Greece, 11 = Austria, 12 = Italy, 13 = Japan, 14 = Netherlands, 15 = United States, 16 = Taiwan

†: A = Aircraft, B = Road traffic, C = Rail traffic, D = Wind turbine, E = Residential Noise, F = Ambient, G = Combined sources of traffic

‡: A = Birth weight, B = Low birth weight, C = Small for gestational age, D = Preterm birth, E = Gestational length, F = Congenital anomalies, G = Very low birth weight, H = Extremely low birth weight, I = Very preterm birth, J = Extremely preterm birth

Table 4: Overview of identified systematic reviews on the impact of environmental noise on the cardiovascular and metabolic system

First author and reference	Studies included								Meta-analysis included
	No evaluated	No of participants	Time range	Countries*	Population$	Noise source(s)†	Setting(s)**	End point(s)‡	
Weihofen, 2019[25]	9	780 – 5,523,788	1947 – Aug 2017	1-10	GP	A	R	D	Yes
Wang, 2020[19]	8	1836 – 380738	2009 – Oct 2019	3, 5, 8, 15, 20, 34	GP	A,B, E	R, O	E	Yes
Fu, 2017[18]	32	59 – 145,190	To Dec 2016	2, 6, 8, 10, 12-21	A	A, B, E	R, O	B	Yes
Sakhvidi, 2018[27]	15	40 – 381,000	To Sept 2017	1, 3, 5, 6, 8, 12, 15, 20, 22- 24	GP	A, B, E, F	R, O	E	Yes
Peters, 2018[22]	17	60 – 4,4 million	2013 - 2017	1, 2, 4-6, 8-10	A	A	R	A, B, C, D, E, G	No
Hadad, 2019[20]	9	420 - ~4.4 million	2007 - 2018	4-6, 8, 9, 20, 25	A	A, B, C	R	B, C, D, G	No
Dzhambov, 2017[16]	13	115 – 1542	To July 2016	1, 4, 6, 7, 14, 17, 21, 26, 27	C	B	R,E	A	Yes
An, 2018[13]	11	132 – 52456	To Feb 2018	1, 8, 10, 20, 23, 27, 28	GP	A, B,C, E, G	R, O, E	H, I	Yes
Munzel, 2017[29]	~19	Up to 8.6 million	NR	1-10, 20, 24	A	A, B, C	R	A, C, D, E, G, H, I	No
Freiberg, 2019[28]	2*	725 – 1238	2000 – Sept 2017	3, 7	GP	D	R	B, C, E	No
Dzhambov, 2018[17]	9	420 – 4,415,206	To August 2017	3-6, 8, 9, 20, 25, 33	A	B	R	B	Yes

Van Kempen, 2018 [5]	61	85 – 6,027,363	2000 – Aug 2015	1-8, 10, 11, 13, 14, 16, 18, 20, 22, 25, 26, 33	A, C	A, B, C, D	R, E	A, B, C, D, E, H, I	Yes
Wilding, 2019 [26]	8 (1)	54,968	1990-Aug 2018	20	C	B, C	R	I, K, N	No
Alves, 2020 [12]	6*	4 – 717453	2016-2019	1, 8, 10, 16, 20	A	B, D, E	R, O	C, D, J	No
Van Kamp, 2020 [23]	18*	420 – 4,400,000	2015-2019	2-6, 8-10, 13, 20, 24, 25, 33	A, C	A, B, C, D	R	A, B, C, D, E, H, I, K	No
Khosravipour, 2020 [21]	13	243-854366	Up to Nov 2019	4, 6, 8, 18, 20	A	B	R	C	Yes
Cai, 2021 [14]	13	6,304 – 4,600,000	2000-2020	2-5, 7, 8, 20, 25	A	A, B, C	R	C, D	Yes
Rugel, 2020 [11]	29[a]	513 – 4,284,680	2003 – 2019	3-10, 16, 20, 23, 25, 33	A	B	R	B, C, D, E, G, M, O, P	No
Dendup 2018 [15]	4[a]	513 – 53673	April 2017	5, 8, 20, 23	A	A, B, C	R	E	No

*Countries: 1 = USA, 2 =France, 3 = Canada, 4 =United Kingdom, 5 = Switzerland, 6 = Germany, 7 = The Netherlands, 8 = Sweden, 9 = Greece, 10 = Italy, 11 = Croatia, 12 = Brazil, 13 = Japan, 14 = Serbia, 15 = Korea, 16 = Taiwan, 17 = Pakistan, 18 = Lithuania, 19 = Iran, 20 = Denmark, 21 = India, 22 = EU, 23 = Bulgaria, 24 = China, 25 = Spain, 26 = Austria, 27 = Slovakia, 28 = Portugal, 29 = Belgium, 30 = Australia, 31 = New Zealand, 32 = Poland, 33 = Norway, 34 = European Union;

† A = Air traffic, B = Road traffic, C = Rail traffic, D = Wind turbines, E = Occupational, F = Recreational, G = Human, ;

‡ A = Blood pressure, B = Hypertension, C = Coronary heart disease (incl myocardial infarction and angina pectoris), D = Stroke/cerebrovascular disease, E = Diabetes, F = Cardiometabolic, G = Heart failure, H = Change in waist circumference, I = Change in BMI, J = Cardiovascular risk factors, K = Obesity, L = Cholesterol, M = Arterial Fibrillation, N = Overweight, O = cardiovascular disease, P = Cardiocirculatroy pathologies; [a] In this review also the impact on other effects was evaluated. These studies were not taken into account for this review. Only the studies dealing with cardiovascular and metabolic effects were considered;

$ Population under investigation: GP =General population, A = Adults, C = Children; ** Setting(s): R = Residence, O = Occupational, E = Educationa

Original studies

Mental health and dementia

We included 28 papers [39-66] that investigated the impact of noise on mental health and dementia (vascular and Alzheimer), other neurodegenerative disorders were not considered. Mental health was assessed mainly using validated questionnaires such as General Health Questionnaire (GHQ-12 [45, 47, 67]), Short Form Health Survey questionnaire (SF 36 [59, 66]), Patient Health Questionnaire (PHQ-9 [65]), or Kessler Psychological distress scale (KS10 [64]. Other measures were redemption of or prescribed medication of antidepressants or anxiolytics [40, 41, 64], Diagnostic Statistical Manual for Mental Disorders (DSM-IV [42, 43]), ICD diagnoses from hospitalization, Health registers or National health insurance registers [48, 50, 63]. Dementia was assessed from ICD diagnoses from hospitalization, Health Registers or National Health Insurance registers [46, 48, 58], by a clinical investigation [61], or by a test battery, neurophysiological examination and MRI [62]. The clearly dominated noise source associated with mental health and dementia was road traffic (20 studies), followed by aircraft and railway (7 studies each). Only a very few studies investigated noise from wind turbines (1), neighborhood (2), and industry (2). Of the included studies, as many as 11 were longitudinal cohort studies, one case control, one ecological time series study, and the remaining 12 were cross sectional. One study evaluated incident hospitalization for depression and other mental disorders within a cohort starting from pregnancy, and two studies investigated the effect of road traffic noise on children, one for the risk of ADHD diagnosis and symptom severity, and the other for the risk of inattention. Mental health was also investigated in one study among adolescents and in one population of young adults. The remaining studies included adults. The outcomes of dementia (Alzheimer, or vascular) were investigated in relation to road noise in six studies, five of those were cohort studies and one ecological time series study.

Findings of the studies:

For hyperactivity/inattention, three studies were included [39, 53, 56]. Traffic noise at residential address or at school was not associated with ADHD symptoms but with a lower risk for ADHD diagnosis: residential RR 0.929/1 dB (95 % CI 0.893, 0.965); school RR 0.94/1dB (95 % CI 0.910, 0.981) [39]. Residual confounders may have affected the lower risk of diagnosis, and the mean levels of traffic noise was modest 52 L_{den}. Weyde, Krog [56], found on the contrary, that traffic noise at the age of 8 was associated with 1.2% increase of parental reported inattention score per 10dB at the age 8. Further, traffic noise levels averaged over 3 to 8 years was associated to a 1.3% point increase per 10 dB inattention at 8 years (average marginal effects from fractional logit models). Traffic noise during pregnancy was also associated to inattention but only for boys, possibly supporting previous studies that exposure during pregnancy may affect gender differently [68]. The third study found that road noise was associated with total behavior problems, however the risk for ADHD was not increased [53]. Noise sensitivity was though significantly associated to internalizing, externalizing and total behavior, especially in the low income group. Previous

reviews on children [33] found based on two studies a positive estimate of RR 1.11 (95 % CI 1.04, 1.19) per 10dB L_{den}, road noise.

Mental health depression and anxiety

Incident cases of depression was reported by two studies [59, 66]. Eze, Foraster [66], found increasing risk for cumulative transportation sources for those exposed to ≥45 L_{den} with 1) single-source, risk RR 1.91 (95 % CI 1.00, 3.63), 2) double-source, RR 1.95, (95 % CI 0.98, 3.84), and 3) triple-source exposures, RR 2.29 (95 % CI, 1.02, 5.14). For single exposures there was a limited association, mainly found for those participants who had not moved between time points of data collection, and aircraft noise. It is possible that the low noise levels in the study with average levels of 35, 55, and 35 L_{den} dB, for aircraft, road and rail respectively, played a role for the lack of association with single sources. Depression was here assessed combining antidepressant medication, physician diagnosis and SF36 <50, which suggests a valid measure of mental health. The importance of how mental health is measured is indicated by another study in the same cohort SAPALDIA [59], where the association of mental health using only SF36 and its subcategories did not detect significant risks with exposures adopting quantile regression models, aircraft: coef 0.89, 95% (CI -0.71, 2.50), road: coef 0.13, (95% CI -0.70, 0.97); and railway: coef -0.76, (95% CI -1.88, 0.36).

Among a large sample of women exposed to road noise during pregnancy, a follow up during 18 years revealed that incident hospitalization for depression and other mental disorders (ICD9; ICD10) were increased [63]. The strongest association was found with L_{night}; HR1.32 (95% CI 1.08–1.63) and HR1.68 (95% CI 1.05–2.67), for L_{night} 60 dB, and L_{night} 70 dB, respectively, compared to L_{night} 50 dB. Interestingly, L_{den} and L_{Aeq} showed less clear associations, indicating that sleep may be a more important moderator than daytime [63].

The exposure modelled at address to aircraft, road and railway traffic noise was compared to cases of hospital diagnosed depression 2006 and 2010 and controls [54].The exposure-risk relationship for road traffic noise was linear, with an increased OR already at 40 to < 45 dB of 1.02 (95% CI 1.00, 1.06), and increasing up to 1.17 (95% CI 1.10, 1.25) for the noise category (≥70 dB). For aircraft noise and rail noise the exposure risk relationship was slightly more inverted u-shaped with for aircraft reaching a maximum OR 1.23 (95% CI 1.19–1.28) at 50–55 dB and decreased at higher categories. Railway noise had a peaked risk estimates at 60–65 dB, OR 1.15, (95 % CI 1.08, 1.22). For aircraft, night time maximum levels of ≥50dB increased depression risk. The highest OR of 1.42 (95% CI 1.33, 1.52) was found for a combined exposure to noise above 50 dB from all three sources.

Incidence of suicides (ICD code X60-X80) from the National Health insurance registers increased within 4 and 8 years follow up, HR 1.32 (95% CI 1.02, 1.70) for the age group 20-54 years, 1.43 (95% CI 1.01, 2.02) for those 55 years and older; and 1.55 (95% CI 1.10, 2.19) for adults with mental illness, with interquartile range of L_{night} from noise from transport, industrial, and recreational activities [50]. Noise levels were measured at representative sites during at least 2 hours at two occasions or when unavailable based on models.

Redemption or prescription of antidepressant or anxiolytic drugs, was analyzed in three studies of which two based the data on registers and one on self-reported use. Only the studies using register data is commented on. Register data on prescribed drugs on anxiolytics, and antidepressants were linked to rail and road traffic noise, as well as greenery and air pollutants [64]. For prescription of anxiolytics but not antidepressants, an OR of 1.07 (95% CI 1.03, 1.11) per IQR increase in road-traffic noise was seen, which was a weaker

association than the odds of greenery and air pollution respectively. The odds for road noise remained after adjustment of NO2 and distance to greenery. Wind turbine noise (WTN), outdoors and indoors, were modelled based on wind turbine type and simulated hourly wind [40]. Levels were calculated as 5 years mean exposure. There was a significant risk for antidepressant redemption and WTN outdoors ≥42 dB (*n*=185) HR 1.17 (95% CI 1.01, 1.35), compared to <24 dB (*n*=60,315). The risk of redemption of antidepressants, related to 5 years mean indoor noise exposure ≥ 15dB LF (*n* = 43) was not significantly increased (HR 0.94, 95% CI 0.70, 1.27) versus <5 dB (*n* = 77,995). The study included a very large population (*n* = 23,393), however very few were exposed to noise levels considered high, making conclusions difficult.

Dementia: Prevalent odds ratios for Alzheimer Disease increased by OR 1.29 per 10 dBA (95% CI 1.08 to 1.55) among participants 65 years or older [61]. Residential noise levels were calculated using geographic covariates based on 5-minute grab samples of A-weighted noise collected at 136 unique locations, collected daytime, on–rush hour periods, with an estimated absolute mean error of 3dBA. No adjustments were made for air pollution, but NO_X levels were not strongly correlated with noise in the region (r = 0.08). Similar estimates of increased risks of developing dementia or cognitive impairment without dementia (CIND) or an HR of 1.3 (95 % CI 1.0, 1.6) per 11.6dB IQR of modelled annual daily traffic noise was found by [62]. When including NO_x, the noise effect was slightly attenuated (HR = 1.2, 95 % CI 0.97, 1.6). The risk of dementia/CIND was elevated when LAeq24h and Lnight noise were higher than 75 and 65 dB respectively.

Yuchi, Sbihi [58] reported that road and highway proximity was associated with Non-Alzheimer Dementia (NAD), as diagnosed from medical health insurance register (HR 1.14, 95% CI 1.07, 1.20), while noise L_{den} exposure was not significantly associated with NAD (HR 1.01, 95% CI 0.99, 1.04). Including noise to a model of air pollution did not change the associations. Greenness suggested protective effects for NAD. Both noise exposure and air pollution were assessed at post code level, introducing exposure misclassification. Similar lack of precision in exposure assessments was present in [48]. Incident cases of dementia and, where specified, Alzheimer's and vascular disease from primary care registers were related to a slight risk increase, HR 1.02, (95 % CI 1.0, 1.05) per IQR L_{night} road noise). HR was reduced to 1.01 (95 % CI 0.98, 1.03) when NO_2 and $PM_{2.5}$ were included in the model.

On the contrary to the above studies, noise was not related to incident cases of dementia recorded during a 15 year follow-up, with a HR 0.95 (95 % CI 0.57, 1.57) [46]. A possible limitation of that study was the range of traffic noise exposure with less than two percent of participants exposed to noise levels ($L_{eq.\ 24\ h}$) equal to or exceeding 60 dB.

In comparing with recent reviews, a meta-analysis by Hegewald, Schubert [7] found that the risk of depression increased by 12% with 10dB L_{den} (95% CI 1.02, 1.23) for aircraft noise. Road and rail noise indicated a small (2-3%) but not statistically significant risk increase. Dzhambov and Lercher [8] reported a tendency to a 4% increase of depression and 12% increase of anxiety for road traffic noise. The review by Dzhambov and Lercher [8] were on the basis of six studies able to provide a linear exposure response relation between depression and L_{den}, ranging from 40 to 76 dB, with a statistically significant increase found from about 55 dB. The exposure response function is still to be decided as also more complex response functions have been indicated in single studies (i.e. [54, 63]).

In the reviews for WHO, dementia was not included, but in the review by Clark, Crumpler [9] they concluded that there was "low quality evidence for no effect of road traffic noise on the incidence of vascular dementia". Evidence was then available from one large-scale UK study which found that the association between road noise and an incidence diagnosis of dementia

became non-significant after adjustment for air pollution [48]. However three longitudinal studies were published in 2020, and thus not included in Clark, Crumpler [9]. Two of these included a large sample and have reasonable good assessment of exposures, and both point to an increased risk of around 30% per 10-11.6 dB increase in traffic noise level, with no adjustment for air pollution. One study had no correlation with NO_x and therefore did not adjust for air pollution, while the HR decreased to 1.2 (95 % CI 0.97, 1.6) after adjustment for NO_x and $PM_{2.5}$. While there is an emerging number of studies, more data is needed, these should include occupational exposures as occupational stress and noise may add to the risk. Also the possibility that noise induced hearing loss or hearing loss in general mediate or moderate the risk of dementia should be taken into account [37].

Studies on birth- and reproductive outcomes including congenital anomalies

We included 15 papers [69-83] that investigated the impact of noise on birth and reproductive outcomes including congenital anomalies. The outcomes were defined as preterm birth (PB <37 weeks), low birth weight (LBW <2500g), all cause still birth, and cause specific stillbirth, birth weight (BW), small for gestational age (SGA, calculated growth curve of weight and gestational age). Further congenital anomalies (during this time period) included diagnosis of congenital heart disease (CHD) [76], polydactylism [82], and congenital hearing dysfunction [78]. Most studies adjusted for SES, mothers smoking and alcohol consumption, ethnicity, marital status, season and year of birth, but not always for parity and only some included mother's chronic illness [72], city effect [74] or occupational exposures i.e. strenuous work, stress, noise or just occupation during pregnancy [79, 81]. If possible, harmonization of confounders among studies would be advised. The clearly dominated noise source investigated was road noise (7 studies), followed by occupational noise (5 studies) and single studies of aircraft, wind turbine and combined sources. For the transportation noises, the exposure levels were derived from modelled estimates. Wind turbine type and simulated hourly wind at each site was used to estimated hourly outdoor and low frequency (LF) indoor WTN at the dwellings of the pregnant women and aggregated as mean nighttime WTN. Of the four original papers on congenital anomalies, three were on occupational noise exposure and only one on environmental exposure (road traffic noise). Occupational noise was assessed using self-reported noise exposure or occupational based exposure estimates, Job Exposure Matrixes (JEM) classifying exposed versus non-exposed mothers. Of the included studies, nine were longitudinal cohort studies, three used registry data, three were case-control and one were cross-sectional.

Findings

Birth weight, premature birth and small for gestational age: Studies report birth outcomes to be associated to several factors apart from known individual maternal and neonatal risk factors. In Yitshak-Sade, Fabian [72] these factors included higher temperature during pregnancy, mothers living in areas with less greenness, living in more walkable areas, and in areas with more of the "low income" population and higher residential noise. Treating the exposures individually, nighttime noise had the highest weight in its contribution to lower birthweight, accounting for 18% of the weights. Single exposure model for each IQR of night time noise of 4.1 dB; -16.88g (95 % CI -18.49, 15.27), and multi-exposure model: -5.63g (95 % CI -7.52; -3.73). This is one of the largest studies today with 640 659 births, however the noise levels were estimated by 270x270 meter grid, providing less well spatial resolution. In a similar direction, it was found that the likelihood of having LBW babies increased by 1.6 percentage points among mothers who lived in the direction of an aircraft runway and were

exposed to more air movements and noise levels over the 55 dB threshold L_{dn}, as compared to those who lived at a further distance [69].

Mixed results were found in two cohorts analyzed by Dzhambov, Markevych [73], both addressed night time residential noise at similar levels (median 52 L_{den}), but used slightly different modelling. One cohort found increased LBW; OR 2.03/10 dB (95 % CI 1.16, 3.54) and the other did not find an association with OR 0.96 (95 % CI 0.66-1.39). Furthermore, the results were mixed also for SGA, but in opposite directions for the two cohorts. Noise levels being assessed 10 years after the birth outcomes and a moderate sample size may affected the possibilities to achieve valid results. Taking the exposome perspective with noise included among 60 exposures, also no association was found between noise and a lower estimate of BW or LBW [74].

Among mothers exposed to urban traffic air pollution and noise (mean L_{Aeq16}, 58dB, L_{night} 53dB), trends of decreasing birth weight with increasing road traffic noise categories were observed, but the effect were strongly attenuated when adjusted for primary traffic related air pollutants, with fine particulars ($PM_{2.5}$ traffic exhaust and $PM_{2.5}$) most consistently affecting LBW and SGA ; LBW night-time ≥65 dB vs <50 dB: OR 1.03, 95 % CI (0.95, 1.11); LBW daytime ≥65 dB vs <55 dB: OR 1.01, (95 % CI 0.95, 1.07); SGA daytime, compared with ≥65 vs <55 dB: OR 1.00, (95 % CI 0.97, 1.04); SGA night-time ≥65 vs <50 dB: OR 1.03, (95 % CI 0.99, 1.08) [75]. The study is one of the larger studies evaluating birth outcomes in relation to traffic noise and air pollution with more than 540 000 births, and used highly spatially resolved air pollution modelling assigned at address level, and noise levels estimated at address point (0.1 dB resolution). A weakness is that adjustment was not done for parity, known to be strongly related to birth weights [84], or for mother's occupation during pregnancy, and that the reference levels for noise comparison were at levels that may still be considered as harmful for sleep and borderline for restoration according to the WHO guidelines [1]. In a follow up study among the same cohort, an elevated risk of preterm birth OR ~1.02, (95 % CI 1.01-1.02) was associated with increasing road traffic noise, but only after adjustment for certain air pollutant exposures, (NO_x and particles) during 1st and 2nd trimester.

Evaluating the possible effect on birth outcome and wind turbine noise, no association was found between WTN and adverse outcomes on LBW, SGA or PTB [80].

Occupational noise exposure was not included in the WHO review [6]. An association between exposure to high ≥85 dBA vs <75 dBA levels of occupational noise for those working throughout the pregnancy (full time workers) (*n* ~ 250 000) and increased SGA OR=1.44 (95% CI 1.01 to 2.03) and similar increase for LBW OR=1.36 (95% CI 1.03 to 1.80), and association for preterm birth <75 vs 75-84 dBA OR=1.13 (95% CI 1.08 to 1.18) was found in a very large study comprising in total 857,010 births [81]. Individual data, occupation, and other occupational exposures were obtained from prenatal care interviews and related to a job exposure matrix (<75dBA; 75-84 dBA; ≥85 dBA). In a smaller sample including both occupation and environmental exposures, Wallas et al [79] found no clear association between maternal occupational noise exposure during pregnancy and low birth weight or prematurity. Unexpectedly however, they did observe an inverse association between maternal road traffic noise exposure during pregnancy and preterm birth, OR 0.72, (95% CI 0.59–0.90 per 10 dB L_{den}. It is possible that residual confounding due to a lack of fully adjustment for SES may be at hand as there was a positive relation between noise levels and higher income.

Congenital anomalies: Overall evidence suggest a tendency to increased risk of congenital anomalies in relation to occupational and environmental noise exposures. However, studies

showing statistically significant associations have performed crude analysis on the effects of occupational noise exposure on specific anomalies. A hospital-based case-control study showed an increased risk of polydactyly among the offspring of mothers working in textile factories exposed to self-reported noise (unadjusted OR 4.89, (95% CI 2.44, 9.81) [82]. Another similar study showed a higher exposure to occupational noise among cases of congenital heart disease, 42.1% vs. 18.3%, $p = 0.012$ [76]. The study investigating the effects of road traffic noise on congenital anomalies could not show statistical significance using comprehensively adjusted analysis. Pedersen, Garne [77] performed adjusted logistic regression analysis with overall statistically insignificant results for the association between road traffic noise and groups of congenital anomalies (e.g., orofacial cleft OR 1.17, (95% CI 0.94, 1.47) and genital anomalies OR 1.13, (95% CI 0.92, 1.37). For the congenital hearing dysfunction, the analysis supported the lack of an association with occupational noise, however researchers were not able to adjust for potential confounders [78]. The literature review on congenital anomalies expands the conclusion of the previous reviews considering the inclusion of other papers not discussed in the previous reviews. The choice of methodological search that focus on the period of exposure and outcome diagnosis instead of on the congenital anomalies term allowed for the inclusion of more studies. Further studies are needed to clarify possible associations between noise and congenital anomalies.

Studies on cardiovascular and metabolic outcomes

Characteristics of the studies

For our overview we identified and selected 44 studies that investigated the impact of one or more sources of transportation noise, or wind turbine noise on one or more cardiovascular and or metabolic end points [46, 48, 79, 85-145]. Furthermore, 15 studies were dealing with the impact of noise on coronary heart disease (CHD) [85, 89, 92, 95, 96, 98-100, 103, 105, 111, 119, 122, 123, 127, 132, 143], while the impact on stroke was addressed in 15 studies [89, 92, 96, 97, 99, 100, 103, 105, 111, 113, 119, 122, 132, 143, 146]. Although studies investigating metabolic endpoints such as diabetes, and indicators such of overweight, are available less often, their number is increasing rapidly. As part of the 44 selected studies, there were also a number of studies which looked at (i) biomarkers and indicators of cardiovascular or metabolic disease (HUNT3, Lifelines, PLOVDIV, DEBATS, SALSA, PIAMA,BGY, KORA, GEONGGI) [88, 102, 109, 110, 121, 129, 130, 140, 142, 145], and (ii) additional cardiovascular end points such as arterial fibrillation and heart failure (DNC, HYENA_GR, SAPALDIA, DCH, WHII, SABRE, SNC, PPS, ONPHEC, EPIC-OXFORD, UK-BIOBANK, SSND-NIVEL, CBS) [89, 92, 95, 96, 108, 111, 119, 122, 126, 132, 133, 135, 143]. Finally, there were also a number of studies, that investigated the impact of noise on children's blood pressure (PIAMA, BAMSE, HELIX) [87, 106, 109], Body Mass Index (BMI) in children or adults (MoBa) (BAMSE) (Slovakia-I) (DNC) (Plovdiv) (HUNT3) (UK-BIOBANK) (Lifelines) (SPALDIA) [66, 79, 93, 107, 112, 142, 147]), Waist Circumference (WC) in children or adults (PIAMA) (SLOVAKIA-I) (DNC) (Plovdiv) (SAPALDIA) (SDPP) (UK-BIOBANK) (Lifelines) (HUNT3) [93, 107, 110, 120, 128, 142, 147]), and other indicators of overweight (e.g. [central] obesity, percentage body fat, waist-hip-ratio). Figure 1 shows for different cardiovascular and metabolic end points what study designs were applied in the studies published during the last ICBEN period covering a period from January 1, 2017 to January 1, 2021. The figure shows that in the selected studies, the association between environmental noise and cardiovascular and metabolic outcomes, was largely investigated by means of cohort studies. For the change in BMI and WC, the selected studies were mostly of cross-sectional design. This is in contrast to e.g. the WHO-review of Kempen,

Casas [5], where for several end points, the design of the studies was largely cross-sectional. It this is an important improvement, since studies with a cross-sectional design have important limitations.

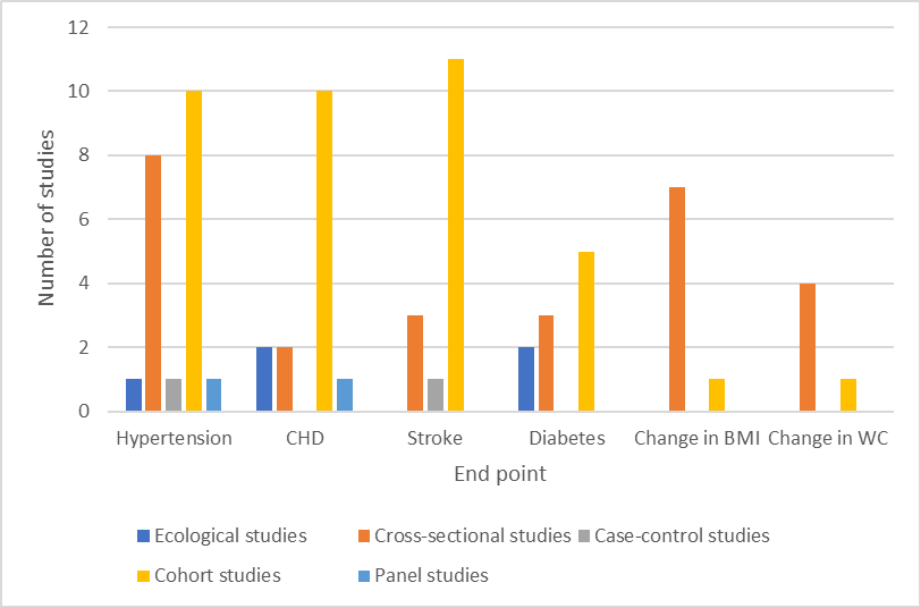

Figure 1: Overview of the different study designs that were in applied in the studies investigating the impact of noise from transportation and wind turbines on hypertension, coronary heart disease (CHD), stroke, diabetes, and change in Body Mass Index (BMI) or Waste Circumference (WC), published during the last ICBEN period.

As expected, road traffic was the most investigated source of noise. The number of studies looking at the impact of rail traffic and wind turbine noise remain limited. Although most of the selected studies, were able to characterize exposure at an individual level, several studies *(WHII, SABRE, ONPHEC, SSND-NIVEL, QICDSS, BASICMAR, or CANADA-1) [85, 90, 95, 97, 111, 133, 144]* were not able to do this. E.g. in the CANADA-1 study [144], the QICDSS study [85], and the ONPHEC study [90, 95], noise exposure was assessed for the postal code in which the participant lived. For the ascertainment of health end points such as the incidence of CHD, or mortality due to stroke, health registries were mostly used. With regard to hypertension, most studies ascertained hypertension by means of a combination of blood pressure measurements and/or self-reported diagnosis for hypertension and/or use of antihypertensive medications (n = 13); also several studies made use of data from health registries, sometimes in combination with blood pressure measurements and self-reported diagnosis of hypertension (n = 4). This is an important improvement in comparison with what was observed in the WHO-review [5], where most studies used only self-reported diagnosis of hypertension. The use of self-reported hypertension can be problematic, since it can lead to underreporting of the outcome. This affects the association with noise, as is amongst others demonstrated by Fuks, Weinmayr [134].

Findings of the studies:

The incidence of hypertension: For the current overview we included 22 studies, investigating the association between noise and hypertension [88, 90, 91, 94, 103, 111, 115, 117, 118, 121, 125, 129, 133, 134, 138, 143]. In 11 studies the association between road traffic noise exposure and the *incidence* of hypertension was investigated: eight cohort studies [88, 89,

115, 134, 143], one case-control study [125] and one ecological study [90]. Furthermore, two meta-analyses [5, 17] were evaluated. The results across the nine cohort studies and case-control study differed in magnitude and direction of the reported effect. This is consistent with the results of the meta-analysis of Dzhambov and Dimitrova [17]. After combining the results of 13 studies, they estimated a RR of 1.02 (95 % CI [0.98, 1.05]) per 10 dB (L_{den}) for the association between road traffic noise and the incidence of hypertension. The findings of Dzhambov and Dimitrova [17] were not consistent with the findings of the WHO review [5], which reported a RR of 0.97 (95% CI [0.90, 1.05]) per 10 dB (L_{den}). This estimate was however based on only one study: the Danish Diet Cancer and Health Cohort (DCH). We evaluated three studies (SDPP, HYENA_GR, NORAH) [115, 125, 143] and one meta-analysis [5] that investigated the impact of air traffic noise. In two of the three studies a positive association was reported. Only the results of the Swedish Stockholm Diabetes Preventive Programm (SDPP) [115] were statistically significant. The power of the NORAH study [125] suffered from the fact that a relatively small number of cases was exposed to high levels of air traffic noise. The results of the HYNEA_GR study were only based on 71 cases. As part of the WHO-review [5], only one cohort study was included investigating the association between air traffic noise exposure and the incidence of hypertension: It were earlier results of the SDPP. In contrast to the recent results [115], no association between aircraft noise and the incidence of hypertension was found. For the current review, we evaluated two studies that investigated the association between rail traffic noise and the incidence of hypertension (SDPP, NORAH) [115, 125]. The results of these studies were inconclusive.

The incidence of CHD: As part of the current overview, we found seven cohort studies [96, 99, 122, 127, 143] and three meta-analyses [5, 21, 122] that reported on the association between road traffic noise exposure and the *incidence* of CHD. The results across these studies differed in the magnitude and direction of the estimated effect: Five of the seven cohort studies found a positive association between road traffic noise exposure and the incidence of CHD; only the results of the DCH study [127] were statistically significant: After adjustment for confounders, a HR of 1.09 (95% CI [1.01, 1.18]) per 10 dB was reported for the association between the mean 1-year exposure to road traffic noise and the incidence of myocardial infarction. As part of their paper on road traffic noise and the incidence of CHD, [122] presented also the results of an update of an earlier meta-analysis, carried out by Vienneau, Schindler [148]. To this end they combined the results of nine studies, revealing a HR of 1.03 (95% CI [1.00, 1.07]) per 10 dB (L_{den}). Recently,CI 1.00 – 1.07) per 10 dB (L_{den}). Recently, Khosravipour and Khanlari [21] published the results of a meta-analysis on the association between road traffic noise and myocardial infarction. After combining the results of 13 studies, a RR of 1.02 (95%CI [1.00 – 1.05]) per 10 dB was estimated. Although the effect estimates derived by. Khosravipour and Khanlari [21] and Cai, Hodgson [122] were smaller, they confirm the results reported by [5]: a RR of 1.08 (95% CI [1.01, 1.15]) per 10 dB (L_{den}). For the current overview, two cohort studies (HYENA_GR and CAENS) [99, 143] were selected that investigated the impact of air traffic noise; no meta-analyses were available that were able to combine results of cohort- or case-control studies investigating the impact of air traffic noise. The results of the two cohort studies were inconclusive. One cohort study (CAENS) [99] also reported on the impact of rail traffic noise: After adjustment for confounders, they reported a HR of 1.01 (95% CI [0.93, 1.09]) per 10 dB L_{den} for the association between the mean 1-year exposure to noise from rail traffic noise and the incidence of CHD. The current overview also included two cohort studies that have investigated the association between noise from wind turbines and the incidence of CHD: The Danish Wind turbine Study (DWS) [100] and the Danish Nurse Cohort (DNC) [123]. After adjustment for confounders, the researchers of the DWS study found that exposure to 1 and

5 year outdoor wind turbine noise levels during the *night* above the reference level (<24 dB L_{night}), was positively but not always significantly associated with myocardial infarction in all exposure groups [100] . In the other Danish cohort study, the DNC study, no associations were found between wind turbine noise (expressed as L_{den}) and the incidence of myocardial infarction [123]. Similar to DWS, no associations were found with wind turbine noise during the night (L_{night}). The power of both the DWS and the DNC study, suffered from the fact that a relatively small number of cases was exposed to high levels of wind turbine noise. Furthermore, the associations in the highest exposure groups were statistically not significant.

Findings of the studies: Mortality due to CHD: For our overview, we identified three cohort studies (SNC, CBS, DCH) [89, 92, 98, 105, 119, 132] and two meta-analyses [5, 14] that investigated the association between noise exposure and mortality due to CHD. In all three cohort studies, the impact of road traffic noise was investigated. In two of the three cohort studies (SNC and DCH), a positive association was found between road traffic noise and mortality due to CHD; in the third study (CBS study), a negative association was reported. Only the results of the Swiss National Cohort (SNC) study were statistically significant. After adjustment for confounders, a HR of 1,023 (95% CI [1,012, 1,034]) per 10 dB (L_{den}) was estimated. On the basis of the results of three studies, Kempen, Casas [5] estimated an RR of 1.05 (95% CI [0.97, 1.13]) per 10 dB (L_{den}). This was consistent with the results of the meta-analysis of [14], who were able to combine the results of six cohort- and case control studies on the association between road traffic noise and mortality due to CHD, and estimated a RR of 1.03 (95% CI [0.98, 1.09]) per 10 dB (L_{den}). In [5], three studies investigating the association between air traffic noise and mortality due to CHD were included: two ecological studies [149, 150] and one cohort study [151]. In the cohort study (earlier results of the SNC-study) a non-significant RR of 1.04 (95%CI [0.98, 1.11]) per 10 dB (L_{den}) was found. For our overview we were able to include one study that investigated the association between air traffic noise and mortality due to CHD: new results of the SNC-study. No association between aircraft noise and mortality due to aircraft noise was found. The new results of the SNC were not consistent with the results reported by Cai et al. (2021) on the association between air traffic noise and mortality due to CHD: for their meta-analysis they included the results of three studies: two ecological studies and one cohort study. From the cohort study they were able to derive an estimate of 1.03 (95 % CI [1.01 -1.05]) per 10 dB (L_{den}). For rail traffic noise, Cai, Ramakrishnan [14] combined the results of two cohort studies, revealing a RR of 1.02 (95 % CI [0.91, 1.14]) per 10 dB L_{den} for the association between rail traffic noise and mortality due to CHD. The results of the two studies on rail traffic included in the current overview (SNC and CBS) were inconsistent. In the WHO review [5] no studies were included that investigated the association between rail traffic noise and mortality due to CHD. For the current review, we did not found any study that investigated the association between noise from wind turbines and mortality due to CHD.

The incidence of stroke: In the current overview, nine studies (eight cohort studies and one case-control study) were included that investigated the association between noise and the *incidence* of stroke [96, 99, 100, 113, 122, 143, 146]. Also two meta-analyses [5, 25] were found that addressed the association between noise and the incidence of stroke. In seven of the nine studies (six cohort studies and one case-control study), the impact of road traffic noise was investigated [96, 99, 113, 122, 143]. The results across these studies differed in the magnitude and direction of the estimated effect: after adjustment for confounders, four of the seven studies found a positive association between road traffic noise exposure and the incidence of stroke. Only in the German NORAH study [113], a statistically significant association was found. After adjustment for a rather limited number of confounders, an OR of

1.017 (95%CI [1.003, 1.032]) per 10 dB ($L_{pAeq24hr}$) was reported. Furthermore, the limitations of the Norah study that were mentioned earlier, were also valid for the association between road traffic noise and the incidence of stroke. The results of the studies in our overview on the association between road traffic noise and the incidence of stroke were not consistent with what was found in the WHO-review [5], including only one cohort study investigating. An RR of 1.14 (95%CI [1.03, 1.25]) per 10 dB (L_{den}) was estimated. Of the nine evaluated studies in our overview, three studies dealt with the association between air traffic noise and the incidence of stroke [99, 113, 143]. The results were not consistent: two studies (the Norah study and the CAENS study), reported a negative association, while one study (HYENA_GR) reported a positive association. However, the results of the latter study were based on only 5 cases. The limitations of the Norah study were already indicated earlier. The results of the current overview are difficult to compare with the results of the meta-analysis of Weihofen, Hegewald [25], since they combined the results of studies with different study designs (including both cross-sectional, cohort- and ecological studies). After combining the results of seven studies, they derived an effect estimate of 1.013 (95%CI: [0.998, 1.028]) per 10 dB (L_{den}). With regard to rail traffic noise, we were able to include the results of two studies stroke [99, 113]. Both found a positive association between rail traffic noise and the incidence of stroke. Only the results of the Norah study were statistically significant: after adjustment for confounders, an OR of 1.018 (95% CI [1.001, 1.034]) per 10 dB ($L_{pAeq24hr}$) was found. None of the meta-analyses [5, 25] reported on the association between rail traffic noise exposure and the incidence of stroke. For the current overview, two cohort studies that investigated the association between wind turbine noise and the incidence of stroke were included: The DNC-study [146] and the DWS-study [100]. In the DWS study, investigating noise exposure during the *night*, IRRs for stroke did not show consistent patterns of associations with 1- and 5-year outdoor wind turbine noise levels during the night. In agreement with the DWS study, the DNC study did not find evidence that long-term exposure to wind turbine noise (expressed as L_{den}) increases the risk of stroke. The earlier mentioned limitations of the DWS- and DNC study, were also valid for the association between noise from wind turbines and the incidence of stroke.

Mortality due to stroke: For the current review, we included three cohort studies (SNC, CBS, and DCH) that investigated the association between road traffic noise and mortality due to stroke [89, 92, 119, 132]. The results of these three studies were consistent: all found a positive, but statistically non-significant association. The results of the two meta-analysis dealing with road traffic noise and stroke, were however inconsistent. On the basis of the results of four cohort studies, Cai, Ramakrishnan [14] estimated a RR of 1.06 (95%CI [0.94, 1.20]) per 10 dB (L_{den}) for the association between road traffic noise and mortality due to stroke. Earlier, the WHO-review reported a RR of 0.87 (95%CI [0.71, 1.06]) per 10 dB (L_{den}) after combining the results of three cohort studies. Both the SNC and CBS study also investigated the association between rail traffic noise and mortality due to stroke [92, 119, 132]. After adjustment for confounders, both studies found no association between rail traffic noise and mortality due to stroke. The SNC study also investigated the impact of air traffic noise. Only a positive and significant association was reported for ischemic stroke: a HR of 1.074 (95%CI [1.020, 1.127]) per 10 dB was estimated. Both Kempen, Casas [5] and Cai, Ramakrishnan [14] did not find an association between air traffic noise and mortality due to stroke on the basis of cohort or case-control studies.

The incidence of diabetes: We evaluated five cohort studies [101, 104, 114, 137, 143] and three meta-analyses [5, 19, 27] that investigated the association between road traffic noise exposure and diabetes. Although the effect sizes of the cohort studies included in the current overview differed between the studies, all found a harmful effect of road traffic noise. In two

studies (SAPALDIA, and DCH) [114, 137] significant effects were found. The results of some of the five studies should however be interpreted with caution, since in some studies (HYENA_GR, SAPALDIA) [137, 143] the number of cases with diabetes was rather small. The results of the meta-analyses were also consistent: In the review of Kempen, Casas [5] a positive and statistically significant association: RR = 1.08 (95%CI [1.02, 1.14]) per 10 dB (L_{den}) was estimated on the basis of one cohort study. H, D [19] was able to combine the results from five publications (describing four studies), revealing an OR of 1.08 (95%CI [1.04, 1.11]) per 10 dB in transportation noise. With regard to air traffic noise, two cohort studies were included in our overview: The HYENA_GR study [143] and the SAPALDIA study [137]. The results were inconclusive. As part of [5], only one cohort study was included that investigated the association between air traffic noise exposure and the incidence of diabetes: the SDPP [152]. No association was found. The current overview included two cohort studies that dealt with the impact of rail traffic noise (DCH and SAPALDIA study) [114, 137]. Both studies found a harmful but statistically not significant effect. As part of [5], only one cohort study (earlier results of the DCH study) [153] was included that investigated the association between rail traffic noise exposure and the incidence of diabetes. Based on the data of this cohort an RR of 0.97 (95%CI: [0.89, 1.05]) per 10 dB (L_{den}) was estimated. The review of Zare Sakhvidi, Zare Sakhvidi [27] also included a meta-analysis dealing with the association between noise and the incidence of diabetes. After combining the results of five cohort studies, they found a RR of 1.04 (95%CI: [1.02, 1.07]) per 5 dB. [27] also combined the results of two case-control studies; these, however, investigated the impact of recreational and occupational noise. For the current overview also one cohort study was included that investigated the impact of noise from wind turbines: The Danish DWS study [116] investigated the impact of noise from wind turbines during the night. No associations were found between wind turbine noise exposure during the night and the incidence of diabetes. These findings were consistent across strata of sex, distance to major road and wind turbine height. As was the case in relation to other outcomes that were investigated in the DWS, there were only a few cases in the highest exposure groups.

GENERAL OBSERVATIONS AND IMPORTANT DEVELOPMENTS

In the studies included in our overview several trends were observed. The most important ones will be discussed shortly.

Noise sources under investigation

For the different health outcomes treated in this overview, road traffic is the source that was mostly investigated. The number of studies on railway noise has increased but are still rare; this also applies to the number of studies of wind turbine noise. However, climate change leads to structural changes, including adaptations linked to energy-saving measures and measures aimed at reduction of CO_2 emissions. Expansion of wind turbine parks, increased freight transport by rail, and the introduction of high speed trains can be identified as direct results of current EU policies regarding adaptive measures. Discussions on health issues related to noise are often neglected but ought to be part of the early process during the decision making on e.g. the siting of wind turbines, the possibilities to reduce the noise impact of high speed trains in the construction phase or the expansion of nighttime freight transport by rail. The number of studies investigating the long-term health impacts of industrial noise, and occupational noise in relation to birth outcomes also remain rare. Even more importantly is that few studies acknowledge the contribution of other noise exposures

such as occupational exposures and noise exposure in the intended restorative green and blue areas.

Populations/population groups under investigation

Most studies that were included in our overview were conducted in developed countries; mostly in the European region. Also, it appeared that most studies were carried out in adult populations, with a handful of papers among children and adolescents within mental health. Studies investigating the impact of noise on the cardiovascular and/or metabolic system were mostly carried out among people in their middle ages and elderly (~40 yrs and older). In contrast to earlier times, studies investigating women or men only, were hardly published (DNC)(PPS) [96, 104, 107, 108, 123, 146].

There is a need for information from countries outside the European region, and among younger subjects. This would improve our understanding of the association between noise and health in a global context and in a life course perspective.

In the last years there has been a growing awareness of the importance of health inequity in the assessment of risks due to environmental exposures. While most studies adjust for indicators of SES (at individual and/or group level), some also adopt a perspective trying to assess the specific vulnerability inherent either due to groups having less resilience due to socio-economic factor or age for example, but also as these individuals may be more vulnerable due to unequal exposure due to poor housing, higher unemployment more dense infrastructure etc. as being observed in [54].

For example: several studies investigating the impact of noise on the cardiovascular and/or metabolic system, have tested whether the association between noise and outcome differed between age groups, between different urbanization areas, between persons with an underlying disease or not (e.g. people with a myocardial infarction or not), between persons exposed to different air pollution exposure groups, etc [92, 104, 107, 108, 115, 117, 120, 123, 135, 137]. There is a need for further studies within this field.

Mechanistic insights: annoyance, noise-sensitivity and sleep

An interesting finding was that annoyance and/or noise sensitivity seemed to mediate the association between noise and mental health [55, 66, 67]. This is biologically plausible as annoyance, in addition to the noise level, may reflect individual variation in noise perception and reaction. Further, noise annoyance triggers negative emotions and activate stress responses in the HPA axis that are involved in the pathophysiology of depression [154]. Noise sensitivity is seen as an indicator of vulnerability to noise and other stressors, and has been related to as a proxy measure of anxiety [155]. This finding seems to be in contrast with the results from cardio-metabolic outcome studies where no interactions between noise and annoyance or and noise and noise-sensitivity was seen [115, 120, 135, 137]. It is though possible that dominating pathways for how noise impact mental health and cardiometabolic outcomes differ, with annoyance being more relevant for mental health and sleep disturbance more relevant for cardiometabolic diseases.

While the importance of sleep for cardiometabolic outcomes are well acknowledged (e.g. interaction between noise and indicator of sleep (quality) [107, 120, 123, 135, 137, 146] and in particular noise disturbed sleep is less often investigated in the case of mental health. Undisturbed sleep may be particularly important for the adolescence period as it is a sensitive periods for brain development of the prefrontal cortex with functions important for planning, attention, working memory, decision making and inhibitory control. However, a bi-

directional relationship between sleep disturbance and dementia has also been indicated among older adults [156].

Exposure-response relations: shapes and thresholds

A lot of the meta-analyses and studies that were included in the current overview were confined to the estimation of a relative risk (expressed as RR, OR, HR, IRR) per 10 dB change in noise level, suggesting an exponential relationship between noise exposure and the prevalence/incidence of the effect concerned. However, this may not always the case; moreover, our overview showed there is still a lot of debate: In several meta-analyses included in the current overview, the authors have investigated whether the shape of the relationship that they observed was linear or not. With regard to the association between road traffic noise and the incidence of CHD, the WHO-review demonstrated the possibility of linear shape, with a threshold somewhere between 50-55 dB (L_{den}) [5]. Both Dzhambov and Dimitrova [17] and Cai, Ramakrishnan [14] found comparable thresholds for road traffic noise and the incidence of hypertension, mortality due to CHD and stroke in their reviews: For the association between road traffic noise and mortality due to CHD, and stroke Cai, Ramakrishnan [14] observed evidence of non-linearity. Visual inspection revealed that for mortality due to CHD the risk started to increase from approximately 53 dB, although such risk was possibly only significant at levels exceeding 55 dB. With regard to road traffic noise and the incidence of hypertension. However, Cai, Ramakrishnan [14] were not able to fully evaluate the shapes of exposure-response relationships for all causes of mortality particularly at higher noise levels (i.e. > 60 dB), as data was limited by the range of noise exposures from available studies. With regard to road traffic noise and the incidence of hypertension, Dzhambov and Dimitrova [17] found not only evidence for a non-linear relationship, but also demonstrated that the risk starts to increase at 50 dB. For the first time, the shape of the association between noise exposure and mental health was investigated [8]. They exact shape of the exposure response function is still to be decided as also more complex response functions have been indicated in single studies (i.e [54, 63]).

Also several *individual* studies provided information about the possible shape of exposure-response -relationships. In several of the individual studies that were included in this overview dealing with cardiometabolic outcomes, the results of categorical analyses were reported. These show a heterogeneous and not always convincing picture. What does not make it easier is the fact that in a number of the studies, the number of participants and number of cases in the highest exposure categories are rather small. And in several studies this is combined with the fact that the risks in the highest exposure categories are usually not statistically significant. A number of the above-described observations were done in cohort studies that were not originally designed to investigate the impact of noise. This poses limitations on the quality control of the exposures and possibilities to draw valid conclusions on associations to health outcomes as limited information is available about contextual, social and personal factors.

Although the exact shape of the relationships and threshold values for the different health outcomes are still under debate, it becomes more and more clear that the risk of noise on serious health effects such as cardiovascular disease or depression starts to increase at lower noise levels than previously thought [1].

New indicators and outcomes

Until recently, much of the research on environmental exposures (such as environmental noise) to date has been largely limited to identifying relationships between individual exposures and single health outcomes. This was also the case for the impact of noise on the cardiovascular system, where most studies were focused on the separate association between different sources of transportation noise on the one hand and outcomes such as blood pressure, hypertension, or CHD (including acute myocardial infarction and angina pectoris) on the other hand. This was also the case during the period 2017-2021. However, during this period also a number of studies appeared that looked into "new" outcomes. As is already indicated in the results section, we observed that more and more studies come available that look at newer health outcomes: e.g. overweight, (central) obesity, arterial fibrillation [96, 108], arrhythmia [143], heart failure [95, 119, 126, 132], depression [66], inattention/ADHD [39, 56], dementia [61, 62].

The same trend can be observed with regard to *early indicators* of e.g. cardiovascular and/or metabolic disease: In addition to classical indicators (pre-cursor) such as blood pressure, more and more studies come available that look into other indicators: indicators of overweight (e.g. change in BMI, change in waist circumference), indicators and/or pre-cursors of cardiovascular disease (e.g. brachial-ankle pulse wave velocity, carotid intima-media thickness), biomarkers of cardiovascular and metabolic disease in blood, serum, saliva or urine (e.g. serum concentrations of high sensitivity C-reactive protein (hsCRP), total cholesterol in serum, triglycerides in serum, glucocorticoids metabolites or the glomerular filtration rate). In addition, a number of studies have started to dig deeper into the mechanisms investigating markers of inflammatory reaction and oxidative stress (i.e. [157, 158]. The studies of new indicators and outcomes are urgently needed to help us in more detail understand the underlying mechanisms between noise and adverse health outcomes.

The health impacts of more than one noise source or combined environmental exposures

Many studies evaluate the impact of several exposures. The most obvious one is air pollution: people living in a city are not only exposed to traffic noise, but also to the air pollution that is generated by this traffic. Several studies have indicated that exposure to air pollution may affect most of the long term health outcomes attributed to transportation noise. Since air pollution and noise from road traffic share the same source, the health impact could be attributed to both exposures. The impact of the natural environment or green and blue areas are frequently evaluated as they are regarded as buffering the noise induces stress by enabling recovery from stress and attention fatigue. Also, during this ICBEN-period several studies have been published that took into account the exposure to air pollutants (e.g. PM_{10}, $PM_{2.5}$, NO_2) and/or the natural environment [44, 58, 74], e.g. (PIAMA) (HUNT3) (Lifelines) (HUNT2) (EPIC-OXFORD) (UK-BIOBANK) (CANADA-1) (DNC) (PLOVDIV) (BAMSE) (SAPALDIA) (DCH) (HNR) (KORA) (SNC) (PHM-2012) (SDPP) (PPS) (ONPHEC). Most studies treated these exposures as a confounder and investigated how the association between noise and the health outcomes changed after additional adjustment for an indicator of air pollution and/or the natural environment. Mixed results were found in a number of studies, the association between noise and an outcome did not change (HUNT2, EPIC-OXFORD, UK-BIOBANK, CANADA-1 DNC PLOVDIV DCH); there was also a group of studies, where the association between noise exposure and outcome clearly weakened [75] (PIAMA) (HUNT3) (Lifelines) (HNR) (KORA) (SNC) (SDPP) (ONPHEC) or increased (SAPALDIA, DCH). In the few studies that investigated the interaction between noise

exposure and air pollution (DNC) (BAMSE) (SAPALDIA) (DCH) (PHM-2012) (SDPP) (PPS) (ONPHEC), only a significant effect was found in the ONPHEC study. Both DCH and SNC study investigated also the effect of combined exposure to noise exposure and air pollution.

Among studies evaluated several exposures a novel finding is that the highest estimate or risk is seen in combined or multi-exposure models. This was for example found for psychological distress and anxiolytics, but not antidepressants where the odds increased from single source models including rail or road noise, to increase with two source models (the inclusion of air pollution $PM_{2.5}$ or NO_2, or index of decreasing green) to become highest in a three source model (noise, air pollution and index of decreasing green) [64]. Of particular interest is that at least two studies find increasing odds when evaluating the combed impact of two or three noise sources [54, 66]. The highest odds found when combining three noise sources (rail, road and aircraft). These findings have implications for how we should evaluate risks for combined exposures, future revisions of exposure response functions but may also be of relevance for risk evaluation of combined environmental and occupational exposures.

Exposome and combined exposures

The exposome may be defined as the totality of environmental exposures from conception onwards [74]. The exposome concept thus evaluate total exposures in a life course perspective which poses many challenges to study design, analyses of associations and analyses of combined exposures. Furthermore, the exposures and its impact on health may have a time dependent relation and the exposure risk can be looked at from at least four perspectives related to risk, where 1) prenatal exposure affects the development of organs during the fetal period and also fetal programming, 2) early age exposure increases the vulnerability for later or latent outcomes in adolescence or adulthood; 3) cumulative exposure affects dose dependent outcomes, and finally, 4) exposure during sensitive or critical periods affects the development of organs or systems. Examples of studies where noise is included are [74, 159]. We foresee a greater number of these studies in the next ICBEN period.

Concluding comment

The above-described developments with regard to new outcomes and indicators, and the impact of exposures other than noise, are important since they not only help us to further elucidate the underlying mechanisms of noise affecting health, but it also fits in to the exposome approach: measuring the totality of environmental exposures that an individual is exposed to across the life course. There is a growing recognition for the need of more complex models to help us understand better how multiple and cumulative environmental exposures affect chronic disease onset, progression and outcomes at critical life stages over the life coarse and across generations and not in the least how we can obtain restorative living and working environments that promotes resilience.

Acknowledgements

Thanks for helpful assistance by fellow members of the Sound Environment and Health research unit www.gu.se/en/research/sound-environment-and-health

REFERENCES

1. *WHO, European noise guidelines for the European region*, W.H.O.R.O.f. Europe, Editor. 2018: Copenhagen, Denmark.
2. Guyatt, G.H., et al., *GRADE: an emerging consensus on rating quality of evidence and strength of recommendations.* 2008. **336**(7650): p. 924-926.
3. Morgan, R.L., et al., *GRADE: Assessing the quality of evidence in environmental and occupational health.* Environ Int, 2016. **92-93**: p. 611-6.
4. Clark, C. and K. Paunovic, *WHO Environmental Noise Guidelines for the European Region: A Systematic Review on Environmental Noise and Quality of Life, Wellbeing and Mental Health.* 2018. **15**(11).
5. Kempen, E.V., et al., *WHO Environmental Noise Guidelines for the European Region: A Systematic Review on Environmental Noise and Cardiovascular and Metabolic Effects: A Summary.* Int J Environ Res Public Health, 2018. **15**(2).
6. Nieuwenhuijsen, M.J., G. Ristovska, and P. Dadvand, *WHO Environmental Noise Guidelines for the European Region: A Systematic Review on Environmental Noise and Adverse Birth Outcomes.* 2017. **14**(10).
7. Hegewald, J., et al., *Traffic Noise and Mental Health: A Systematic Review and Meta-Analysis.* 2020. **17**(17).
8. Dzhambov, A.M. and P. Lercher, *Road Traffic Noise Exposure and Depression/Anxiety: An Updated Systematic Review and Meta-Analysis.* 2019. **16**(21).
9. Clark, C., C. Crumpler, and H. Notley, *Evidence for environmental noise effects on health for the United Kingdom policy context: A systematic review of the effects of environmental noise on mental health, wellbeing, quality of life, cancer, dementia, birth, reproductive outcomes, and cognition.* 2020. **17**(2).
10. Dzhambov, A.M. and P. Lercher, *Road Traffic Noise Exposure and Birth Outcomes: An Updated Systematic Review and Meta-Analysis.* 2019. **16**(14).
11. Rugel, E.J. and M. Brauer, *Quiet, clean, green, and active: A Navigation Guide systematic review of the impacts of spatially correlated urban exposures on a range of physical health outcomes.* Environ Res, 2020. **185**: p. 109388.
12. Alves, J.A., et al., *Low-frequency noise and its main effects on human health-A review of the literature between 2016 and 2019.* Applied Sciences (Switzerland), 2020. **10**(15).
13. An, R., et al., *Chronic Noise Exposure and Adiposity: A Systematic Review and Meta-analysis.* American Journal of Preventive Medicine, 2018. **55**(3): p. 403-411.
14. Cai, Y., R. Ramakrishnan, and K. Rahimi, *Long-term exposure to traffic noise and mortality: A systematic review and meta-analysis of epidemiological evidence between 2000 and 2020.* Environmental Pollution, 2021. **269**.
15. Dendup, T., et al., *Environmental Risk Factors for Developing Type 2 Diabetes Mellitus: A Systematic Review.* Int J Environ Res Public Health, 2018. **15**(1).
16. Dzhambov, A.M. and D.D. Dimitrova, *Children's blood pressure and its association with road traffic noise exposure – A systematic review with meta-analysis.* Environmental Research, 2017. **152**: p. 244-255.

17. Dzhambov, A.M. and D.D. Dimitrova, *Residential road traffic noise as a risk factor for hypertension in adults: Systematic review and meta-analysis of analytic studies published in the period 2011–2017.* Environmental Pollution, 2018. **240**: p. 306-318.
18. Fu, W., et al., *Association between exposure to noise and risk of hypertension: A meta-analysis of observational epidemiological studies.* Journal of Hypertension, 2017. **35**(12): p. 2358-2366.
19. H, W., et al., *Association between noise exposure and diabetes: meta-analysis.* Environmental Science and Pollution Research, 2020. **27**: p. 36085 - 36090.
20. Hahad, O., et al., *The Cardiovascular Effects of Noise.* Dtsch Arztebl Int, 2019. **116**(14): p. 245-250.
21. Khosravipour, M. and P. Khanlari, *The association between road traffic noise and myocardial infarction: a systematic review and meta-analysis.* Science of the Total Environment, 2020. **731**: p. 193226.
22. Peters, J.L., et al., *Aviation Noise and Cardiovascular Health in the United States: a Review of the Evidence and Recommendations for Research Direction.* Curr Epidemiol Rep, 2018. **5**(2): p. 140-152.
23. van Kamp, I., et al., *Evidence relating to environmental noise exposure and annoyance, sleep disturbance, cardio-vascular and metabolic health outcomes in the context of IGCB (N): A scoping review of evidence regarding sources other than transport noise*. 2020, National Institute for Public Health and the Environment, Centre for Sustainability, Environment and Health, PoBox1, Postbus 10, BA rivm, 3280, Netherlands UK Department for the Environment, Food and Rural Affairs (Defra), Ground Floor, Seacole Building, 2 Marsham Street, London, SW1P 4DF, United Kingdom Netherlands Institute for Health Services Research, Utrecht, 3513 CR, Netherlands.
24. van Kamp, I. *Update of the WHO evidence reviews and their implications for policy and research*. 2020. Australian Acoustical Society.
25. Weihofen, V.M., et al., *Aircraft noise and the risk of stroke - A systematic review and meta-analysis.* Deutsches Arzteblatt International, 2019. **116**(14): p. 237-244 and I.
26. Wilding, S., et al., *Maternal and early-life area-level characteristics and childhood adiposity: A systematic review.* Obes Rev, 2019. **20**(8): p. 1093-1105.
27. Zare Sakhvidi, M.J., et al., *Association between noise exposure and diabetes: A systematic review and meta-analysis.* Environmental Research, 2018. **166**: p. 647-657.
28. Freiberg, A., et al., *Health effects of wind turbines on humans in residential settings: Results of a scoping review.* 2019. **169**: p. 446-463.
29. Münzel, T. and M. Sørensen, *Noise pollution and arterial hypertension.* European Cardiology Review, 2017. **12**(1): p. 26-29.
30. Zare Sakhvidi, F., et al., *Environmental Noise Exposure and Neurodevelopmental and Mental Health Problems in Children: a Systematic Review.* 2018. **5**(3): p. 365-374.
31. Zare Sakhvidi, F., et al., *Correction to: Environmental Noise Exposure and Neurodevelopmental and Mental Health Problems in Children: a Systematic Review.* 2018. **5**(3): p. 396.
32. Robbins, R.N., et al., *Impact of urbanization on cognitive disorders.* 2019. **32**(3): p. 210-217.

33. Schubert, M., et al., *Behavioral and Emotional Disorders and Transportation Noise among Children and Adolescents: A Systematic Review and Meta-Analysis.* 2019. **16**(18).
34. Dickerson, A.S., et al., *A Scoping Review of Non-Occupational Exposures to Environmental Pollutants and Adult Depression, Anxiety, and Suicide.* 2020. **7**(3): p. 256-271.
35. Mucci, N., et al., *Urban noise and psychological distress: A systematic review.* International Journal of Environmental Research and Public Health, 2020. **17**(18): p. 1-22.
36. Peris, E. and B. Fenech, *Associations and effect modification between transportation noise, self-reported response to noise and the wider determinants of health: A narrative synthesis of the literature.* 2020. **748**: p. 141040.
37. Huang, L., et al., *Relationship Between Chronic Noise Exposure, Cognitive Impairment, and Degenerative Dementia: Update on the Experimental and Epidemiological Evidence and Prospects for Further Research.* 2021. **79**(4): p. 1409-1427.
38. Lan, Y., et al., *Transportation noise exposure and anxiety: A systematic review and meta-analysis.* 2020. **191**: p. 110118.
39. Zijlema, W.L., et al., *Associations between road traffic noise exposure at home and school and ADHD in school-aged children: the TRAILS study.* 2021. **30**(1): p. 155-167.
40. Poulsen, A.H., et al., *Impact of Long-Term Exposure to Wind Turbine Noise on Redemption of Sleep Medication and Antidepressants: A Nationwide Cohort Study.* 2019. **127**(3): p. 37005.
41. Rudolph, K.E., et al., *Environmental noise and sleep and mental health outcomes in a nationally representative sample of urban US adolescents.* 2019. **3**(4): p. e056.
42. Generaal, E., et al., *Neighbourhood characteristics and prevalence and severity of depression: pooled analysis of eight Dutch cohort studies.* Br J Psychiatry, 2019. **215**(2): p. 468-475.
43. Generaal, E., et al., *Not urbanization level but socioeconomic, physical and social neighbourhood characteristics are associated with presence and severity of depressive and anxiety disorders.* Psychol Med, 2019. **49**(1): p. 149-161.
44. Dzhambov, A., et al., *Urban residential greenspace and mental health in youth: Different approaches to testing multiple pathways yield different conclusions.* 2018. **160**: p. 47-59.
45. Dzhambov, A.M., et al., *Multiple pathways link urban green- and bluespace to mental health in young adults.* 2018. **166**: p. 223-233.
46. Andersson, J., et al., *Road traffic noise, air pollution, and risk of dementia - results from the Betula project.* Environmental Research, 2018. **166**: p. 334-339.
47. Baudin, C., et al., *Aircraft Noise and Psychological Ill-Health: The Results of a Cross-Sectional Study in France.* 2018. **15**(8).
48. Carey, I.M., et al., *Are noise and air pollution related to the incidence of dementia? A cohort study in London, England.* 2018. **8**(9): p. e022404.
49. Ma, J., et al., *A Multilevel Analysis of Perceived Noise Pollution, Geographic Contexts and Mental Health in Beijing.* 2018. **15**(7).
50. Min, J.Y. and K.B. Min, *Night noise exposure and risk of death by suicide in adults living in metropolitan areas.* 2018. **35**(9): p. 876-883.

51. Okokon, E.O., et al., *Traffic noise, noise annoyance and psychotropic medication use.* 2018. **119**: p. 287-294.
52. Wright, D.M., et al., *Aircraft noise and self-assessed mental health around a regional urban airport: a population based record linkage study.* 2018. **17**(1): p. 74.
53. Lim, J., et al., *Negative impact of noise and noise sensitivity on mental health in childhood.* Noise & health, 2018. **20**(96): p. 199-211.
54. Seidler, A., et al., *Association between aircraft, road and railway traffic noise and depression in a large case-control study based on secondary data.* 2017. **152**: p. 263-271.
55. Park, J., et al., *Noise sensitivity, rather than noise level, predicts the non-auditory effects of noise in community samples: a population-based survey.* 2017. **17**(1): p. 315.
56. Weyde, K.V., et al., *Road traffic noise and children's inattention.* Environmental Health, 2017. **16**(1): p. 127.
57. Linares, C., et al., *Short-term association between environmental factors and hospital admissions due to dementia in Madrid.* 2017. **152**: p. 214-220.
58. Yuchi, W., et al., *Road proximity, air pollution, noise, green space and neurologic disease incidence: a population-based cohort study.* 2020. **19**(1): p. 8.
59. Cerletti, P., et al., *The independent association of source-specific transportation noise exposure, noise annoyance and noise sensitivity with health-related quality of life.* Environment International, 2020. **143**.
60. Ma, J., et al., *Assessing personal noise exposure and its relationship with mental health in Beijing based on individuals' space-time behavior.* 2020. **139**: p. 105737.
61. Weuve, J., et al., *Long-term community noise exposure in relation to dementia, cognition, and cognitive decline in older adults.* 2020.
62. Yu, Y., et al., *Traffic-related Noise Exposure and Late-life Dementia and Cognitive Impairment in Mexican-Americans.* 2020. **31**(6): p. 771-778.
63. He, S., et al., *Residential noise exposure and the longitudinal risk of hospitalization for depression after pregnancy: Postpartum and beyond.* 2019. **170**: p. 26-32.
64. Klompmaker, J.O., et al., *Associations of combined exposures to surrounding green, air pollution and traffic noise on mental health.* 2019. **129**: p. 525-537.
65. Leijssen, J.B., et al., *The association between road traffic noise and depressed mood among different ethnic and socioeconomic groups. The HELIUS study.* 2019. **222**(2): p. 221-229.
66. Eze, I.C., et al., *Incidence of depression in relation to transportation noise exposure and noise annoyance in the SAPALDIA study.* Environment International, 2020. **144**.
67. Dzhambov, A.M., et al., *Pathways linking residential noise and air pollution to mental ill-health in young adults.* 2018. **166**: p. 458-465.
68. Hines, M., et al., *Prenatal stress and gender role behavior in girls and boys: a longitudinal, population study.* 2002. **42**(2): p. 126-134.
69. Argys, L.M., S.L. Averett, and M. Yang, *Residential noise exposure and health: Evidence from aviation noise and birth outcomes.* 2020. **103**.
70. Smith, R.B., et al., *Impacts of air pollution and noise on risk of preterm birth and stillbirth in London.* 2020. **134**: p. 105290.

71. Wandschneider, L., et al., *Small-Area Factors and Their Impact on Low Birth Weight-Results of a Birth Cohort Study in Bielefeld, Germany.* 2020. **8**: p. 136.
72. Yitshak-Sade, M., et al., *Estimating the Combined Effects of Natural and Built Environmental Exposures on Birthweight among Urban Residents in Massachusetts.* 2020. **17**(23).
73. Dzhambov, A.M., I. Markevych, and P. Lercher, *Associations of residential greenness, traffic noise, and air pollution with birth outcomes across Alpine areas.* 2019. **678**: p. 399-408.
74. Nieuwenhuijsen, M.J., et al., *Influence of the Urban Exposome on Birth Weight.* 2019. **127**(4): p. 47007.
75. Smith, R.B., et al., *Impact of London's road traffic air and noise pollution on birth weight: retrospective population based cohort study.* 2017. **359**: p. j5299.
76. Gong, W., et al., *Congenital heart defects of fetus after maternal exposure to organic and inorganic environmental factors: a cohort study.* 2017. **8**(59): p. 100717-100723.
77. Pedersen, M., et al., *Exposure to air pollution and noise from road traffic and risk of congenital anomalies in the Danish National Birth Cohort.* 2017. **159**: p. 39-45.
78. Guven, S.G., et al., *Does noise exposure during pregnancy affect neonatal hearing screening results?* 2019. **21**(99): p. 69-76.
79. Wallas, A., et al., *Traffic noise exposure in relation to adverse birth outcomes and body mass between birth and adolescence.* 2019. **169**: p. 362-367.
80. Poulsen, A.H., et al., *Pregnancy exposure to wind turbine noise and adverse birth outcomes: a nationwide cohort study.* 2018. **167**: p. 770-775.
81. Selander, J., et al., *Full-time exposure to occupational noise during pregnancy was associated with reduced birth weight in a nationwide cohort study of Swedish women.* 2019. **651**: p. 1137-1143.
82. Shi, J., et al., *Maternal occupational exposure to chemicals in the textile factory during pregnancy is associated with a higher risk of polydactyly in the offspring.* J Matern Fetal Neonatal Med, 2019: p. 1-7.
83. Barba-Vasseur, M., et al., *Does low to moderate environmental exposure to noise and air pollution influence preterm delivery in medium-sized cities?* Int J Epidemiol, 2017. **46**(6): p. 2017-2027.
84. Shah, P.S., *Parity and low birth weight and preterm birth: a systematic review and meta-analyses.* Acta Obstet Gynecol Scand, 2010. **89**(7): p. 862-75.
85. Yankoty, L.I., et al., *Manuscript title: Long—term residential exposure to environmental/transportation noise and the incidence of myocardial infarction.* International Journal of Hygiene and Environmental Health, 2021. **232**.
86. Thacher, J.D., et al., *Road traffic noise exposure and filled prescriptions for antihypertensive medication: A danish cohort study.* Environmental Health Perspectives, 2020. **128**(5).
87. Warembourg, C., et al., *Urban environment during early-life and blood pressure in young children.* Environment International, 2021. **146**.
88. Yu, Y., et al., *Air pollution, noise exposure, and metabolic syndrome – A cohort study in elderly Mexican-Americans in Sacramento area.* Environment International, 2020. **134**.
89. Thacher, J.D., et al., *Long-term residential road traffic noise and mortality in a Danish cohort.* Environmental Research, 2020. **187**.

90. Shin, S., et al., *Association Between Road Traffic Noise and Incidence of Diabetes Mellitus and Hypertension in Toronto, Canada: A Population-Based Cohort Study.* Journal of the American Heart Association, 2020. **9**(6): p. e013021.
91. Nguyen, T.L., et al., *Effects of changes in acoustic and non-acoustic factors on public health and reactions: Follow-up surveys in the vicinity of the hanoi noi bai international airport.* International Journal of Environmental Research and Public Health, 2020. **17**(7).
92. Klompmaker, J.O., et al., *Surrounding green, air pollution, traffic noise exposure and non-accidental and cause-specific mortality.* Environment International, 2020. **134**.
93. Cai, Y., et al., *Impact of road traffic noise on obesity measures: Observational study of three European cohorts.* Environmental Research, 2020. **191**.
94. Baudin, C., et al., *The role of aircraft noise annoyance and noise sensitivity in the association between aircraft noise levels and hypertension risk: Results of a pooled analysis from seven European countries.* Environmental Research, 2020. **191**.
95. Bai, L., et al., *Exposure to road traffic noise and incidence of acute myocardial infarction and congestive heart failure: A population-based cohort study in Toronto, Canada.* Environmental Health Perspectives, 2020. **128**(8): p. 1-9.
96. Andersson, E.M., et al., *Road traffic noise, air pollution and cardiovascular events in a Swedish cohort.* Environmental Research, 2020. **185**.
97. Vivanco-Hidalgo, R.M., et al., *Association of residential air pollution, noise, and greenspace with initial ischemic stroke severity.* Environmental Research, 2019. **179**.
98. Vienneau, D., et al., *Façades, floors and maps – Influence of exposure measurement error on the association between transportation noise and myocardial infarction.* Environment International, 2019. **123**: p. 399-406.
99. Pyko, A., et al., *Long-term transportation noise exposure and incidence of ischaemic heart disease and stroke: A cohort study.* Occupational and Environmental Medicine, 2019. **76**(4): p. 201-207.
100. Poulsen, A.H., et al., *Long-term exposure to wind turbine noise and risk for myocardial infarction and stroke: A nationwide cohort study.* Environmental Health Perspectives, 2019. **127**(3).
101. Ohlwein, S., et al., *Indoor and outdoor road traffic noise and incident diabetes mellitus: Results from a longitudinal German cohort study.* Environmental Epidemiology, 2019. **3**(1).
102. Lee, P.J., et al., *Association between transportation noise and blood pressure in adults living in multi-storey residential buildings.* Environment International, 2019. **132**.
103. Klompmaker, J.O., et al., *Associations of combined exposures to surrounding green, air pollution, and road traffic noise with cardiometabolic diseases.* Environmental Health Perspectives, 2019. **127**(8).
104. Jørgensen, J.T., et al., *Long-term exposure to road traffic noise and incidence of diabetes in the danish nurse cohort.* Environmental Health Perspectives, 2019. **127**(5).
105. Héritier, H., et al., *A systematic analysis of mutual effects of transportation noise and air pollution exposure on myocardial infarction mortality: A nationwide cohort study in Switzerland.* European Heart Journal, 2019. **40**(7): p. 598-603.

106. Enoksson Wallas, A., et al., *Traffic noise and other determinants of blood pressure in adolescence.* International Journal of Hygiene and Environmental Health, 2019. **222**(5): p. 824-830.
107. Cramer, J., et al., *Road traffic noise and markers of adiposity in the Danish nurse cohort: A cross sectional study.* Environmental Research, 2019. **172**: p. 502-510.
108. Bräuner, E.V., et al., *Long-term wind turbine noise exposure and the risk of incident atrial fibrillation in the Danish Nurse cohort.* Environment International, 2019. **130**.
109. Bloemsma, L.D., et al., *Green space, air pollution, traffic noise and cardiometabolic health in adolescents: The PIAMA birth cohort.* Environment International, 2019. **131**.
110. Bloemsma, L.D., et al., *The associations of air pollution, traffic noise and green space with overweight throughout childhood: The PIAMA birth cohort study.* Environ Res, 2019. **169**: p. 348-356.
111. Zock, J.P., et al., *The impact of social capital, land use, air pollution and noise on individual morbidity in Dutch neighbourhoods.* Environment International, 2018. **121**: p. 453-460.
112. Weyde, K.V., et al., *A longitudinal study of road traffic noise and body mass index trajectories from birth to 8 Years.* Epidemiology, 2018. **29**(5): p. 729-738.
113. Seidler, A.L., et al., *The effect of aircraft, road, and railway traffic noise on stroke - results of a case-control study based on secondary data.* Noise & health, 2018. **20**(95): p. 152-161.
114. Roswall, N., et al., *Long-term exposure to residential railway and road traffic noise and risk for diabetes in a Danish cohort.* Environmental Research, 2018. **160**: p. 292-297.
115. Pyko, A., et al., *Transportation noise and incidence of hypertension.* International Journal of Hygiene and Environmental Health, 2018. **221**(8): p. 1133-1141.
116. Poulsen, A.H., et al., *Long-term exposure to wind turbine noise at night and risk for diabetes: A nationwide cohort study.* Environmental Research, 2018. **165**: p. 40-45.
117. Poulsen, A.H., et al., *Long-term exposure to wind turbine noise and redemption of antihypertensive medication: A nationwide cohort study.* Environment International, 2018. **121**: p. 207-215.
118. Michaud, D.S., et al., *Clarifications on the Design and Interpretation of Conclusions from Health Canada's Study on Wind Turbine Noise and Health.* Acoustics Australia, 2018. **46**(1): p. 99-110.
119. Héritier, H., et al., *Diurnal variability of transportation noise exposure and cardiovascular mortality: A nationwide cohort study from Switzerland.* International Journal of Hygiene and Environmental Health, 2018. **221**(3): p. 556-563.
120. Foraster, M., et al., *Long-term exposure to transportation noise and its association with adiposity markers and development of obesity.* Environment International, 2018. **121**: p. 879-889.
121. Carugno, M., et al., *Effects of aircraft noise on annoyance, sleep disorders, and blood pressure among adult residents near the Orio al Serio International Airport (BGY), Italy.* La Medicina del lavoro, 2018. **109**(4): p. 253-263.

122. Cai, Y., et al., *Road traffic noise, air pollution and incident cardiovascular disease: A joint analysis of the HUNT, EPIC-Oxford and UK Biobank cohorts.* Environment International, 2018. **114**: p. 191-201.
123. Bräuner, E.V., et al., *Long-term wind turbine noise exposure and incidence of myocardial infarction in the Danish nurse cohort.* Environment International, 2018. **121**: p. 794-802.
124. Andersen, Z.J., et al., *Long-term exposure to road traffic noise and incidence of myocardial infarction. a Danish nurse cohort study.* Occupational and Environmental Medicine, 2018. **75**: p. A20.
125. Zeeb, H., et al., *Traffic noise and hypertension – results from a large case-control study.* Environmental Research, 2017. **157**: p. 110-117.
126. Sørensen, M., et al., *Long-term exposure to road traffic noise and nitrogen dioxide and risk of heart failure: A cohort study.* Environmental Health Perspectives, 2017. **125**(9).
127. Roswall, N., et al., *Long-term residential road traffic noise and NO2exposure in relation to risk of incident myocardial infarction – A Danish cohort study.* Environmental Research, 2017. **156**: p. 80-86.
128. Pyko, A., et al., *Long-Term Exposure to Transportation Noise in Relation to Development of Obesity—a Cohort Study.* Environmental health perspectives, 2017. **125**(11): p. 117005.
129. Pitchika, A., et al., *Long-term associations of modeled and self-reported measures of exposure to air pollution and noise at residence on prevalent hypertension and blood pressure.* Science of the Total Environment, 2017. **593-594**: p. 337-346.
130. Lefèvre, M., et al., *Effects of aircraft noise exposure on saliva cortisol near airports in France.* Occupational and Environmental Medicine, 2017. **74**(8): p. 612-618.
131. Kim, A., et al., *Effects of self-reported sensitivity and road-traffic noise levels on the immune system.* PLoS ONE, 2017. **12**(10).
132. Héritier, H., et al., *Transportation noise exposure and cardiovascular mortality: a nationwide cohort study from Switzerland.* European Journal of Epidemiology, 2017. **32**(4): p. 307-315.
133. Halonen, J.I., et al., *Associations of night-time road traffic noise with carotid intima-media thickness and blood pressure: The Whitehall II and SABRE study cohorts.* Environment International, 2017. **98**: p. 54-61.
134. Fuks, K.B., et al., *Long-termexposure to ambient air pollution and traffic noise and incident hypertension in seven cohorts of the European study of cohorts for air pollution effects (ESCAPE).* European Heart Journal, 2017. **38**(13): p. 983-990.
135. Foraster, M., et al., *Exposure to road, railway, and aircraft noise and arterial stiffness in the SAPALDIA study: Annual average noise levels and temporal noise characteristics.* Environmental Health Perspectives, 2017. **125**(9).
136. Eze, I.C., et al., *Exposure to night-time traffic noise, melatonin-regulating gene variants and change in glycemia in adults.* International Journal of Environmental Research and Public Health, 2017. **14**(12).
137. Eze, I.C., et al., *Long-term exposure to transportation noise and air pollution in relation to incident diabetes in the SAPALDIA study.* International Journal of Epidemiology, 2017. **46**(4): p. 1115-1125.

138. Evrard, A.S., et al., *Does aircraft noise exposure increase the risk of hypertension in the population living near airports in France?* Occupational and Environmental Medicine, 2017. **74**(2): p. 123-129.
139. Dzhambov, A.M., et al., *Community Noise Exposure and its Effect on Blood Pressure and Renal Function in Patients with Hypertension and Cardiovascular Disease.* Folia medica, 2017. **59**(3): p. 344-356.
140. Dzhambov, A.M., et al., *Is community noise associated with metabolic control in patients with cardiovascular disease?* Accoustics Australia, 2017. **45**: p. 61-75.
141. Dzhambov, A.M. and D.D. Dimitrova, *Road traffic noise-related hypertension and ethnicity as an effect modifier*, in *Traffic Noise: Exposure, Health Effects and Mitigation*. 2017. p. 41-58.
142. Dzhambov, A.M., et al., *Association between community noise and adiposity in patients with cardiovascular disease.* Noise & health, 2017. **19**(91): p. 270-277.
143. Dimakopoulou, K., et al., *Is aircraft noise exposure associated with cardiovascular disease and hypertension? Results from a cohort study in Athens, Greece.* Occupational and Environmental Medicine, 2017. **74**(11): p. 830-837.
144. Clark, C., et al., *Association of long-term exposure to transportation noise and traffic-related air pollution with the incidence of diabetes: A prospective cohort study.* Environmental Health Perspectives, 2017. **125**(8).
145. Cai, Y., et al., *Long-termexposure to road traffic noise, ambient air pollution, and cardiovascular risk factors in the HUNTand lifelines cohorts.* European Heart Journal, 2017. **38**(29): p. 2290-2296.
146. Bräuner, E.V., et al., *Association Between Long-Term Exposure to Wind Turbine Noise and the Risk of Stroke: Data From the Danish Nurse Cohort.* Journal of the American Heart Association, 2019. **8**(14).
147. Argalášová, Ľ., et al. *Is Transportation Noise Associated with Obesity?* 2017.
148. Vienneau, D., et al., *The relationship between transportation noise exposure and ischemic heart disease: a meta-analysis.* Environ Res, 2015. **138**: p. 372-80.
149. Hansell, A.L., et al., *Aircraft noise and cardiovascular disease near Heathrow airport in London: small area study.* 2013. **347**: p. f5432.
150. Van Poll R. and et al., *Gezondheidsonderzoek Vliegbasis Geilenkirchen (Desk Research) I. Hoofdrapportage: samenvatting, conclusies en aanbevelingen Gezondheidsonderzoek Vliegbasis Geilenkirchen (in Dutch)*. 2014, RIVM: Bilthoven.
151. Huss, A., et al., *Aircraft noise, air pollution, and mortality from myocardial infarction.* Epidemiology, 2010. **21**(6): p. 829-36.
152. Eriksson, C., et al., *Long-term aircraft noise exposure and body mass index, waist circumference, and type 2 diabetes: a prospective study.* Environ Health Perspect, 2014. **122**(7): p. 687-94.
153. Sørensen, M., et al., *Long-term exposure to road traffic noise and incident diabetes: a cohort study.* Environ Health Perspect, 2013. **121**(2): p. 217-22.
154. Keller, J., et al., *HPA axis in major depression: cortisol, clinical symptomatology and genetic variation predict cognition.* Mol Psychiatry, 2017. **22**(4): p. 527-536.
155. Stansfeld, S.A. and M. Shipley, *Noise sensitivity and future risk of illness and mortality.* Sci Total Environ, 2015. **520**: p. 114-9.

156. Mander, B.A., J.R. Winer, and M.P. Walker, *Sleep and Human Aging.* Neuron, 2017. **94**(1): p. 19-36.
157. Eze, I.C., et al., *Genome-wide DNA methylation in peripheral blood and long-term exposure to source-specific transportation noise and air pollution: The SAPALDIA study.* Environmental Health Perspectives, 2020. **128**(6).
158. Münzel, T., et al., *Environmental stressors and cardio-metabolic disease: Part II-mechanistic insights.* European Heart Journal, 2017. **38**(8): p. 557-564.
159. Maitre, L., et al., *Early-life environmental exposure determinants of child behavior in Europe: A longitudinal, population-based study.* Environment International, 2021. **153**: p. 106523.